DISSERTATION ON

"A COMPARATIVE STUDY OF DYNAMIC MRI WITH DRUG INDUCED SLEEP ENDOSCOPY IN OBSTRUCTIVE SLEEP APNEA PATIENTS."

Dissertation submitted in partial fulfillment of the regulations for the award of the degree of

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BONAFIDE CERTIFICATE

This is to certify that this dissertation is a bonafide record of work done by Dr.R.CHANDRU, on **"A COMPARATIVE STUDY OF DYNAMIC MRI WITH DRUG INDUCED SLEEP ENDOSCOPY IN OBSTRUCTIVE SLEEP APNEA PATIENTS"** during his M.S. ENT course from April 2013 to April 2016 at the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai. He is appearing for his M.S. Branch – IV Degree Examination in April –2016 and his work has been done with partial fulfilment of the regulations of The TamilNadu Dr.M.G.R Medical University, Chennai. I forward this to The TamilNadu Dr. M.G.R Medical University, Chennai, TamilNadu, India.

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DECLARATION

I, **DR.R.CHANDRU**, solemnly declare that this dissertation entitled **"A COMPARATIVE STUDY OF DYNAMIC MRI WITH DRUG INDUCED SLEEP ENDOSCOPY IN OBSTRUCTIVE SLEEP APNEA PATIENTS."**is a bonafide work done by me in Upgrade Institute Of Otorhinolaryngology, Madras Medical College and Rajiv Gandhi General Hospital, Chennai during the period of 2013 to 2016 under the guidance of **Prof.Dr.M.K.RAJASEKAR M.S.D.L.O.,** Professor, Institute Of Otorhinolaryngology, Madras Medical College and Rajiv Gandhi General Hospital, Chennai – 3 and submitted to The Tamilnadu Dr.M.G.R. Medical University, Guindy, Chennai – 32 in the partial fulfillment of the regulations for the award of the M.S.E.N.T ., (Branch IV).

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Place :Chennai.
Date :

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ABBREVIATIONS

OSAS	:	obstructive sleep apnea syndrome
OBST	:	obstruction
DISE	:	Drug induced sleep endoscopy
ITH	:	Inferior turbinate hypertrophy
EPS	:	Epworth Sleep Scale
PSG	:	Polysomnography
AHI	:	Apnea-Hypopnea Index
TFT	:	Thyroid Function Test
CPAP	:	Continuous Positive Airway pressure
BMI	:	Body Mass Index
UPPP	:	Uvulopalatopharyngoplasty
TBR	:	Tongue Base Reduction
PP	:	Palatoplasty / Palatal Reduction
Antr	:	Anterior
Postr	:	Posterior
m	:	muscle
PSG	:	Polysomnography
AHI	:	Apnea-Hypopnea Index
TFT	:	Thyroid Function Test
CPAP	:	Continuous Positive Airway Pressure
BMI	:	Body Mass Index
UPPP	:	Uvulopalatopharyngoplasty
TBR	:	Tongue Base Reduction
PAP	:	Positive airway pressure

MMP	:	Modified Mallampati Index
SDB	:	Sleep Disordered Breathing
AASM	:	American Academy Of Sleep Medicine
SL	:	Sleep Latency
TST	:	Total Sleep Time
SE	:	Sleep Efficiency
TIB	:	Total time in bed
RAS	:	Reticular activating system
ACH	:	Acteylcholine
PGE2	:	Prostaglandin E2
GABA	:	Gamma amino butyric acid
HMS	:	Hyoid myotomy with suspension
MMP	:	Maxillo-mandibular advancement procedures

CONTENTS

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a component of sleep disordered breathing and this disorder is characterized by excessive snoring and periodic apneas, hypopneas and arousals that leads to fragmented sleep in a repetitive specific duration .OSAS is a disease of modern ages and identified as distinct entity for past 20 to 25 years .At present ,OSAS has been identified as a separate risk factor or an entity for increased susceptibility to stroke, myocardial infarction, cardiac arrhythmias, hypertension, dyslipidemia, insulin resistance and diabetes mellitus, depression, sexual dysfunction .Impairment of alertness also increases the risk of susceptible patients to occupational hazards and automobile accidents.

Sleep is a " transient state of altered consciousness and perceptual disengagement from one's surrounding environment". More over the sleep phenomenon is a active process associated with a profound physiological alterations involving a complex interactions and processing among various parts of brain especially cortical and diencephalic structures. Under normal circumstances, physiological systemic functions associated with sleep occur without any serious consequences. However in pathological states, the changes ensue in any of these systemic functions may present serious physiological risks with consequences that affect the qualitative and quantitative aspects of sleep and daytime functions .Henceforth majority of this renewed interest within the otolaryngologists has been focused on sleep related breathing disorder OSA and this recognition has led to a

Multi disciplinary approach with a creation of new medical discipline – Sleep medicine; with a teams made up of otolaryngologists, pulmonologists ,neurologists, maxillofacial surgeons, and behavioral psychologists.

HISTORICAL PERSPECTIVES

Day time somnolence associated with obesity was first described by "CHARLES DICKENS" in 1837 through his writing in "The Posthumous Papers of the Pickwick club"

1956 – Drs.A.G.Bicklemann, C.S.Burwell ,and colleagues described "Pickwickian Syndrome" .

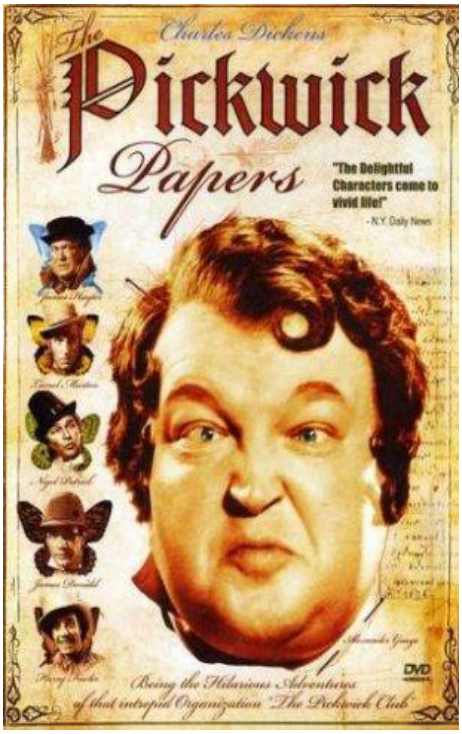
1970 –Elio Lugaresi's described completely about "OSA syndrome".

1983 – Riley described cephalometric evaluation for OSA.

1985 – Fujita described "Uvulopalatopharyngoplasty (UPPP)".

1986 – Colin Sullivan ,a young Australian medical researcher developed nasal "Continuous Positive Airway Pressure".

1990-dr.murray john developed ESS



Charles Dickens

CLASSIFICATION OF OBSTRUCTIVE SLEEP-RELATED BREATHING DISORDERS;

SNORING

Defined as “sound generated by the vibration of pharyngeal soft tissues”. Usually it is more during inspiration than expiration; and it may also present as a separate symptom without any association to day time sleepiness.

UPPER AIRWAY RESISTANCE SYNDROME:

UARS is a recent entity which “describes patients with symptoms of OSA and PSG evidence of sleep fragmentation with AHI<5 without oxyhemoglobin saturation”

OBSTRUCTIVE SLEEP APNEA SYNDROME

OSA is defined as “five or more respiratory events (apneas,hypopneas, or RERAs) in association with excessive daytime somnolence,waking with

gasping, choking, or breath-holding, or witnessed reports of apneas, loud snoring or both".

RESPIRATORY EVENTS DEFINITIONS

APNEA

"Cessation of airflow for at least 10 seconds".

HYPOPNEA

"Reduction in airflow (≥30%) at least 10 seconds with ≥4% Oxy-hemoglobin desaturation or reduction in airflow (≥50%) at least 10 seconds with ≥3% oxy-hemoglobin desaturation or an EEG arousal".

RESPIRATORY EFFORT-RELATED AROUSAL (RERAS)

"Sequence of breaths for at least 10 seconds with increasing respiratory effort or flattening of nasal pressure waveform leading to an arousal from sleep when the sequence of breaths does not meet the criteria for an apnea or hypopnea".

APNEA-HYPOPNEA INDEX

It is calculated as "number of apneas and hypopneas per hour of total sleep time".

Normal	:	AHI<5
Mild OSA	:	AHI 5-15
Moderate OSA	:	AHI 16-30
Severe OSA	:	AHI>30"

OBESITY HYPOVENTILATION SYNDROME

It is defined as “combination of obesity (BMI>30Kg/m2) ,hypoxemia during sleep,hypercapnia during daytime resulting from hypoventilation”

TYPES OF APNEA

OBSTRUCTIVE

It is expressed as “respiratory event with continued thoracoabdominal effort in the setting of partial or complete airflow cessation”.

CENTRAL

It is the “lack of thoraco abdominal effort in the setting of partial or complete airflow cessation”.

MIXED

“This usually begins as central events and end up with a thoraco - abdominal effort in cessation of airflow”. Hence it is mixture of Both central and obstructive respiratory events.

CENTRAL APNEA INDEX

“Number of central apneas per hour of total sleep time”.

MIXED APNEA INDEX

Expressed as “number of mixed apneas per hour of total sleep time”.(1)

ANATOMY OF SNORERS AND APNEIC PATIENTS

In OSA patients, there is collapse of different pharyngeal soft tissue structures especially that of velopharynx, oropharynx, and /or hypopharynx

inaddition to soft palate vibrations .Based on the different sites of pharyngealcollapse, “OSA patients are structurally classified as:

1) Type-1: narrowing or collapse in the retropalatal (velopharyngeal) region alone

2) Type-2: narrowing or collapse in both retropalatal and retroglossal regions .

3) Type-3: narrowing or collapse in the retroglossal region alone”.

PHYSIOLOGY OF SLEEP

Sleep is a temporary state of unconsciousness that can be interrupted by external stimuli and it is regulated by RAS.

THEORIES OF SLEEP

PASSIVE THEORY

Discharge from RAS during prolonged hours of wakefulness leaads to fatigue of RAS thereby inducing sleep.

ACTIVE THEORY

1) Serotonin from raphe fibres inhibits RAS therby promoting sleep

2) Melatonin from pineal gland inhibits reticular activating system thereby promoting sleep

NEUROTRANSMITTERS INHIBITING SLEEP

1) Histamine acts on posterior hypothalamus to promote alertness and wakefulness thereby inhibiting sleep.

2) Aspartate and glutamate acts on thalamo cortical neurons leading to cortical activation thereby promoting sleep.

3) ACH/GABA acts on telencephalon leading to wakefulness.

4) PGE2 release causes wakefulness.(2)

SLEEP PROMOTING NEUROTRANSMITTERS:

1) Serotonin inhibits posterior hypothalamus and RAS and promotes sleep.

2) Melatonin inhibits suprachiasmatic nucleus to promote sleep.

3) PGD2 released from medial preoptic area of hypothalamus promotes sleep.

4) Increased adenosine concentration has found to promote sleep.

GENESIS OF NREM SLEEP

NREM Sleep is produced by stimulation of three subcortical centers

1) “Low frequency stimulation of diencephalic sleep zone in posterior hypothalamus and anterior thalamic nucleus promotes NREM sleep”

2) “Low frequency stimulation of medullary synchronizing zone in NTS promotes NREM sleep.

3) Either low or high frequency stimulation of basal forebrain(preoptic area and diagonal band of broca)promote sleep.(2)

GENESIS OF REM SLEEP

"PGO spikes due to discharge from cholinergic neurons in lateral pontine tegmentum promotes REM sleep" "During REM sleep there is increased activity in pontine area, amygdala, anterior cingulate gyrus and visual association areas and decreased activity in prefrontal ,parietal cortex and primary visual cortex."

EEG WAVES

ALPHA WAVES

- "Awake but at rest with mind wandering, eyes closed
- 8-12HZ
- 50-100 UV
- Parieto occipital area"

BETA WAVES

- "18-30HZ
- Amplitude lower
- Frontal Region"

THETA WAVES

- "Hippo campus
- 4-7 HZ
- Amplitude large"

DELTA WAVES

- “<4HZ
- Amplitude large”

STAGES OF SLEEP

During normal sleep, a young adult first enters NREM sleep, asses through stages 1&2 spends 70-100 min in stages 3&4,sleep then lightens and passes to REM sleep. His cycle is repeated at intervals of every 90 min. But towards morning time there is more of REM sleep and less of stage 3&4 sleep.

STAGE-1

- “4-5 % (Light Sleep)
- Low Amplitude
- High Frequency
- Muscle Activity
- Slows Down
- Transition From alpha wave to theta waves”.

STAGE-2

- “ 45-55 % (Breathing Pattern)
- HR Slows
- Theta Activity

- Sleep spindles & K complexes”

STAGE-3

- “4-6 % (Deep Sleep)
- Delta wave
- Slow High amplitude
- Low frequency”

STAGE-4

- “ 12-15 % (Very Deep Sleep)
- Muscle Activity decreases
- Delta Waves
- Large marked synchronization.”

REM SLEEP

- “20-25%
- Rapid, low voltage EEG activity
- Paradoxical sleep
- PGO Spickes
- Hypotonicity
- Dreams
- 80%in premature infants
- 50% in children
- 25% in adults

	REM	NREM
Duration	20-25%	75-80%
Eye Mvt	Rapid	No
Autonomic activity	Increased fluctuation in BP,HR,RR	Decreased Low BP, slow HR, Steady respiration
Brain Activity	Active	Minimal
Muscular activity	Decreased	Functional but less
EEG	Low voltage	Passes from alpha to delta
Dreaming	Yes	No

OBSTRUCTIVE SLEEP-DISORDERED BREATHING

STATIC AND DYNAMIC FORCES

It is easy when conceptualizing the upper airway to a oversimplified complex interactions, but when dividing it into various forces based on static and dynamic components it gives a better concepts on correlation between sleep and breathing. The static determinants of airway size are contributed by the anatomical structures especially the intrinsic pharyngeal area mainly constructed by the craniofacial framework and upper airway soft tissue mass.Whereas the dynamic forces are contributed by phasic neuromuscular tone and dynamic airflow .And each of these forces have additional levels of controls, complexity ,physiology and pathology .

Anatomical abnormalities including smaller maximal upper airway, increased compliance as the airway decreases in size ,more positive closing

pressures, and increased airway length are the known abnormal static features responsible for sleep disordered breathing. These abnormal static characteristics result in an abnormally collapsible air-column on exposure to the conditions of dynamic flow.

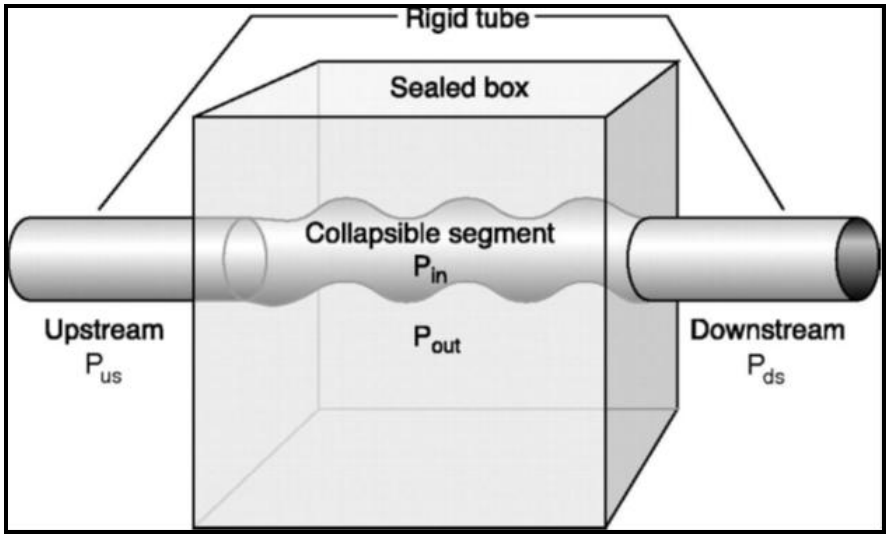
The mechanics of upper airway collapse may be described using both a static model that evaluates the changes due to independent airflow system and also a dynamic model that evaluates the changes in respect to negative inspiratory pressure and airflow .Classically ,it was thought that the uppe rairway collapse occurs only during the inspiration when negative inspiratory pressure and airflow predominate .However the models state that collapse is not only limited to inspiration but also occurs during expiration. This critical event of expiratory collapse occurs when static characteristics predominate.

BALANCE OF FORCES AND STARLING RESISTOR

Integrating the multitude of anatomical or static forces and physiological processes or dynamic forces into a manageable pattern is done using these models.

Balance of forces explains about the multiple forces acting on theupper airway system leading to their collapse or altering its size.These collapsing forces include tissue mass, surface adhesive forces, and negative intraluminal pressures.

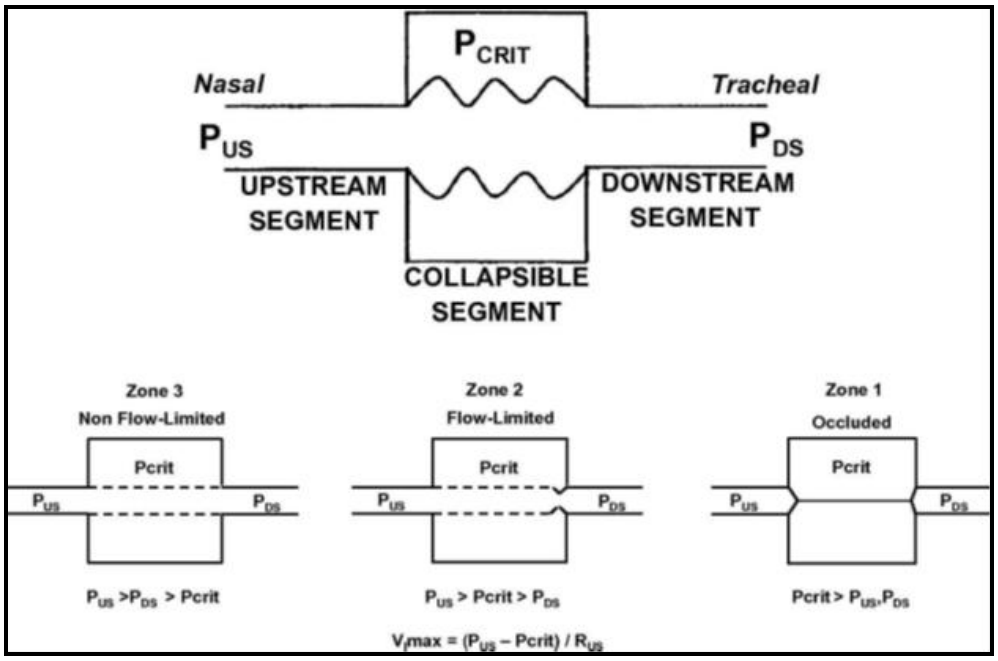
“Ptm = Pin – Pout = Ptissue – Pluminal”



Starling resistor is builded upon the concept of “Poiseuille’s law”, and it describes the pattern and effect of airflow in collapsible tubes resemblingthe upper airway system.

“ V = P1-P2 / R”

Three basic clinical patterns namely normal breathing, snoring, and obstruction are explained by this method.



ANATOMY OF SOFT PALATE (VELUM):

It is a mobile musculo aponeurotic connective tissue suspended from posterior part of hard palate. The lateral part of soft palate is continuous with palatoglossal and palate pharyngeal folds. Soft palate has two layers of mucosa between which there is a fibrous band called palatine aponeurosis which is expanded tendon of m.tensorveli palate.(3)

During deglutition soft palate is made taut which makes the tongue to press against the soft palate thereby allowing food bolus to squeezed into oral cavity. Then soft palate is elevated postero-superiorly to press against the posterior pharyngeal wall by the action of m.levatorveli palate to prevent food entering nasal cavity.

MUSCLES OF SOFT PALATE

- m.tensor veli palate
- m.levatorveli palate
- m.uvulae
- m.palatoglossus
- m.palatopharyngeus

TENSOR VELI PALATI

It originates from “medial pterygoid plate of sphenoid, spine of sphenoid, cartilage of auditory tube” and it gets inserted to palatine aponeurosis . It’s main action is tensor of soft palate and opens auditory tube while swallowing and yawning.(4)

LEVATOR VELI PALATI

It orginates from “cartilage of auditory tube and inferior surface of petrous part of temporal bone”, gets inserted into palatine aponeurosis. it’s main action is elevation of soft palate.

PALATOGLOSSUS

It originates from palatine aponeurosis and gets inserted to side of tongue.it’s main action is elevation of posterior part of tongue and approximates soft palate and tongue.

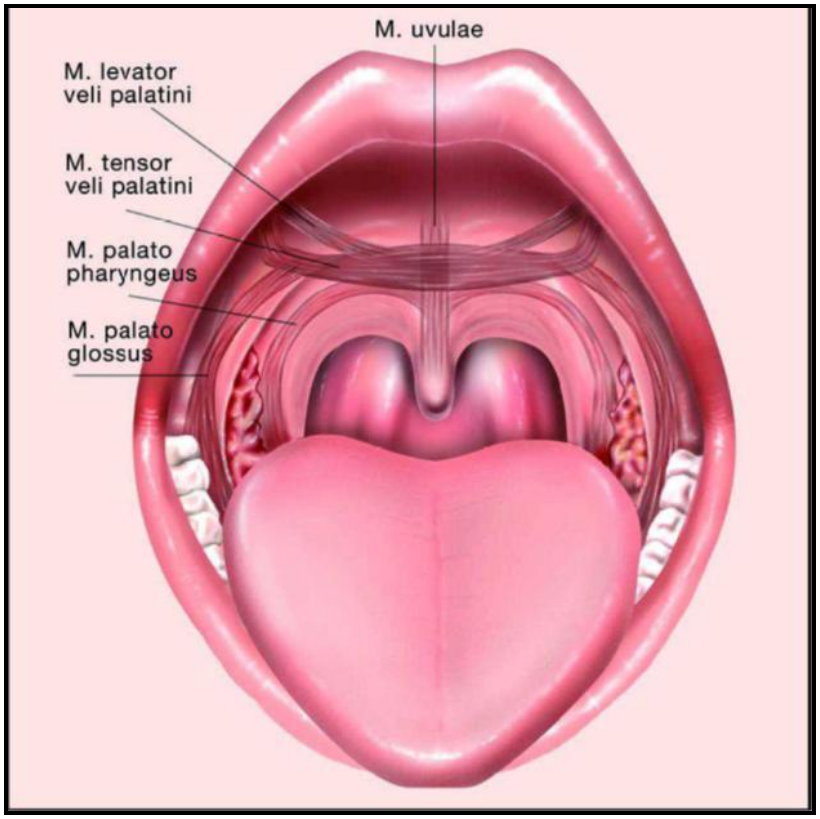
PALATOPHARYNGEUS

It originates from palatine aponeurosis and gets inserted into lateral wall of pharynx.it elevates the pharyngeal wall antero superiorly and medially during deglutition.

MUSCULUS UVULAE

It originates from palatine aponeurosis and gets inserted into mucosa of uvula. It acts to pull the uvula up and shorten it. It is the only intrinsic muscle of larynx.

“All muscles of soft palate are innervated by pharyngeal plexus except m.tensorveli palate which is innervated by trigeminal nerve.”



PALATOPHARYNGEAL SPHINCTER(PASSAVANT'S MUSCLE);

It arises from anterosuperior surface of palatine aponeurosis to blend with upper border of superior pharyngeal constrictor to encircle the pharynx as a sphincter.it acts along with palatopharyngeus and levator palate to help in closure of pharyngeal isthmus. (3,4).

MUSCLES OF TONGUE

INTRINSIC MUSCLES

1) Superior longitudinal muscles-make dorsal surface of tongue concave and shorten the length of tongue.

2) Inferior longitudinal muscles-make dorsal surface of tongue convex and shorten the length of tongue.

3) Transversus linguae-reduce the width and increase the length of tongue.

4) Verticalis linguae-increase the width of the tongue and make the dorsal surface concave from side to side.

EXTRINSIC MUSCLES

1) Styloglossus-pulls tongue upwards and backwards.

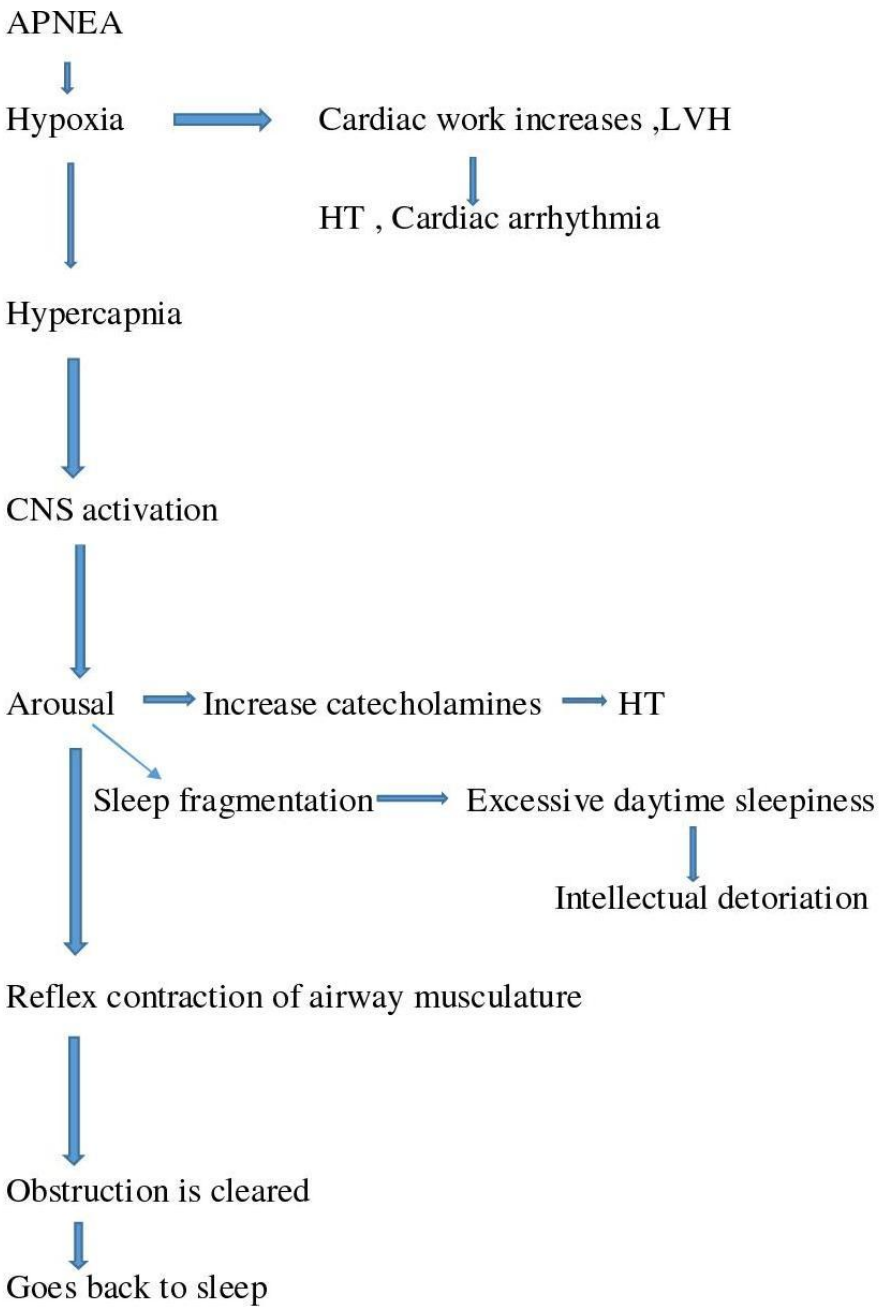
2) Palatoglossus-muscles of both side acting together bring the palatoglossal arches together closing the aperture from oral cavity to pharynx.

3) Genioglossus-protrudes tongue (safety muscle of tongue)

4) Hyoglossus-depresses tongue

"Allmuscles of tongue are supplied by hypoglossal nerve except palatoglossus which is supplied by cranial part of accessory nerve".(4)

PATHOPHYSIOLOGY OF OSA



The etiology and mechanism of airway collapse and OSA is Multi factorial and it is largely due to the interaction of collapsible upper airway with the relaxation of the pharyngeal dilator muscles. Notably, patients without anatomical abnormalities may also present with OSA. Obesity, soft

tissue hypertrophy , and craniofacial characteristics increases the propensity for OSA by increasing the extra luminal tissue pressures. The three major areas of obstruction are the nose, the palate and the hypopharynx . Nasal obstruction mainly contributes for increased airway resistance. At present, obesity is considered as an independent and major risk factor for sleep disordered breathing. Obesity contributes to OSA mainly through two pathological processes namely the mechanical effect of tracheal and thoracic traction called as 'tracheal tug' and by the hypertrophy of genioglossus muscle, which is considered to be the most important muscle in maintaining the upper airway in OSA patients .(5)

OSAS affects 2-5% of population.40-60 years is the most commonest age group affected. Males are commonly affected(M:F=2:1).Post menopausal women have 2-3 fold increased risk of developing OSAS. Obesity and weight gain are the two most important risk factors for development of OSAS. It is a gradually progressive disease characterized by palatal denervation with a localized polyneuropathy and inflammatory cell infiltration of soft palate caused by snoring related vibrations and large intra luminal pressure differences due to obstruction.

SYMPTOMS OF OSAS

NOCTURNAL SYMPTOMS

1) "Snoring

2) Witnessed apneas

3) Dyspnea

4) Drooling

5) Dry mouth

6) Bruxism

7) Restless sleep/frequent arousals

8) Gastroesophageal reflux

9) Nocturia”(6)

DAYTIME SYMPTOMS

1) “Excessive day time sleepiness

2) Morning headaches

3) Neuro cognitive impairment

4) Diminished quality of life

5) Mood and personality changes

6) Sexual dysfunction”.(6)

COMMON FINDINGS IN OSAS

1) Craniofacial:

2) “Retrognathia

3) High arched palate

4) TMJ dislocation”(6)

5) Pharyngeal:

6) “Macroglossia

7) Erythema/edema of uvula

8) Elongated,low lying soft palate

9) Tonsillar pillar hypertrophy

10) Tonsillar enlargement

11) Retropalatal,retroglossal space restriction”(6)

DENTAL

- “Overjet
- Malocclusion
- Bruxism
- Orthodontia”

NASAL

- “Asymmetric small nares
- Inspiratory collapse of alae and internal valves
- Septal deviation
- Inferior turbinate hypertrophy”

EVALUATION OF OSAS

POLYSOMNOGRAPHY

Investigation should be considered if the patient presents with persistent snoring, and at least one other associated symptom. “excessive daytime sleepiness, Impaired cognitive function -difficulty concentrating, depression, learning and memory difficulties, personality changes, and hyperactivity in children.

Morning headaches, decreased libido and impotence in men impair work performance Cardiovascular- hypertension and insulin resistance, heart attack, cardiac arrhythmia, and stroke”.(7)

PSG is the‘‘gold standard’’ for diagnosing SDB and other sleep disorders.It allows “qualitative and quantitative documentation of abnormalities of sleep and wakefulness, sleep-wake transition, and of physiological function of other organ systems that are influenced by sleep.”(7)

INDICATIONS OF PSG

1) Diagnosing SDB and its treatment with CPAP

2) Evaluation for effectiveness of alternative treatments for SDB (eg, dental appliances or surgical procedures)

3) Diagnosis of sleep-related seizures and parasomnias

4) Evaluation of erectile dysfunction with nocturnal penile tumescence [NPT]).(7)

TYPES OF PSG

Level I-“Standard PSG with minimum of seven parameters measured (EEG,EOG, chin EMG,ECG, airflow,respiratory effort, and oxygen saturation) in a specilist unit”.

Level II-“Comprehensive portable PSG same,except that a heart rate monitor can replace the ECG and a technician is not in constant attendance”

Level III.-“Modified portable sleep apnea testing is a cardiorespiratory study ,minimum of four parameters ventilation (at least two channels of respiratory movement, or respiratory movement and airflow), heart rate or ECG, and oxygen saturation”. Done at home.

Level IV-“Continuous (single or dual) bioparameter recordings where devices that measure a minimum of one parameter, usually oxygen saturation are utilized”(7)

Additional variables that can be measured with PSG are Body position,

end-tidal carbon dioxide monitoring (EtCO2), 16-channel EEG recording for seizures, esophageal pressure monitoring (Pes),pulse transit time (PTT) and videography of the whole session can be taken.

A major disadvantage in this technique is that ,breathing disturbances may vary from night to night within certain limits .Some sleep disorders such as nocturnal laryngospasm, sleep related epilepsy, and parasomnias that occur episodically may be missed in one-night measurement .

SLEEP HYPNOGRAM

A sleep hypnogram is a summary of the entire night's PSG data in a graphic form. It gives a good snapshot of sleep architecture, distribution of respiratory events, and oxygen saturation trends in different sleep stages,sleep position, and at different times of the study night. It is helpful to open a window for the hypnogram simultaneously while reviewing the PSG.

The most important parameters measured in sleep hypnogram are:

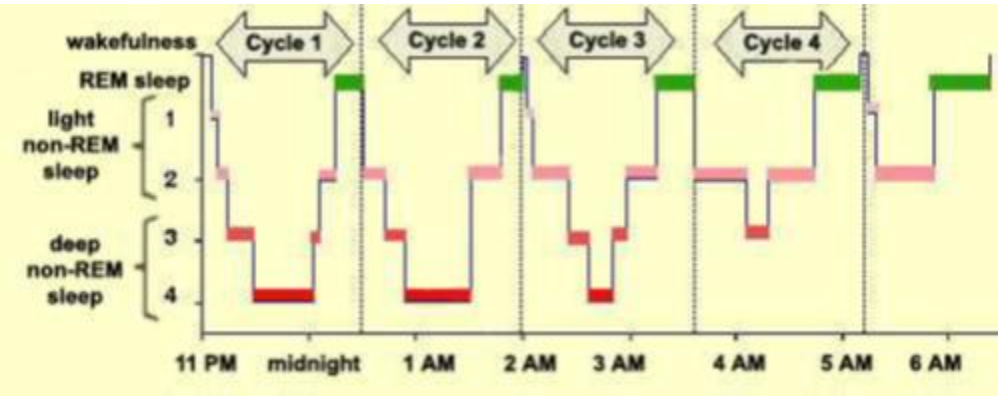
1) Total recording time(TRT)-" beginning and end of recording"

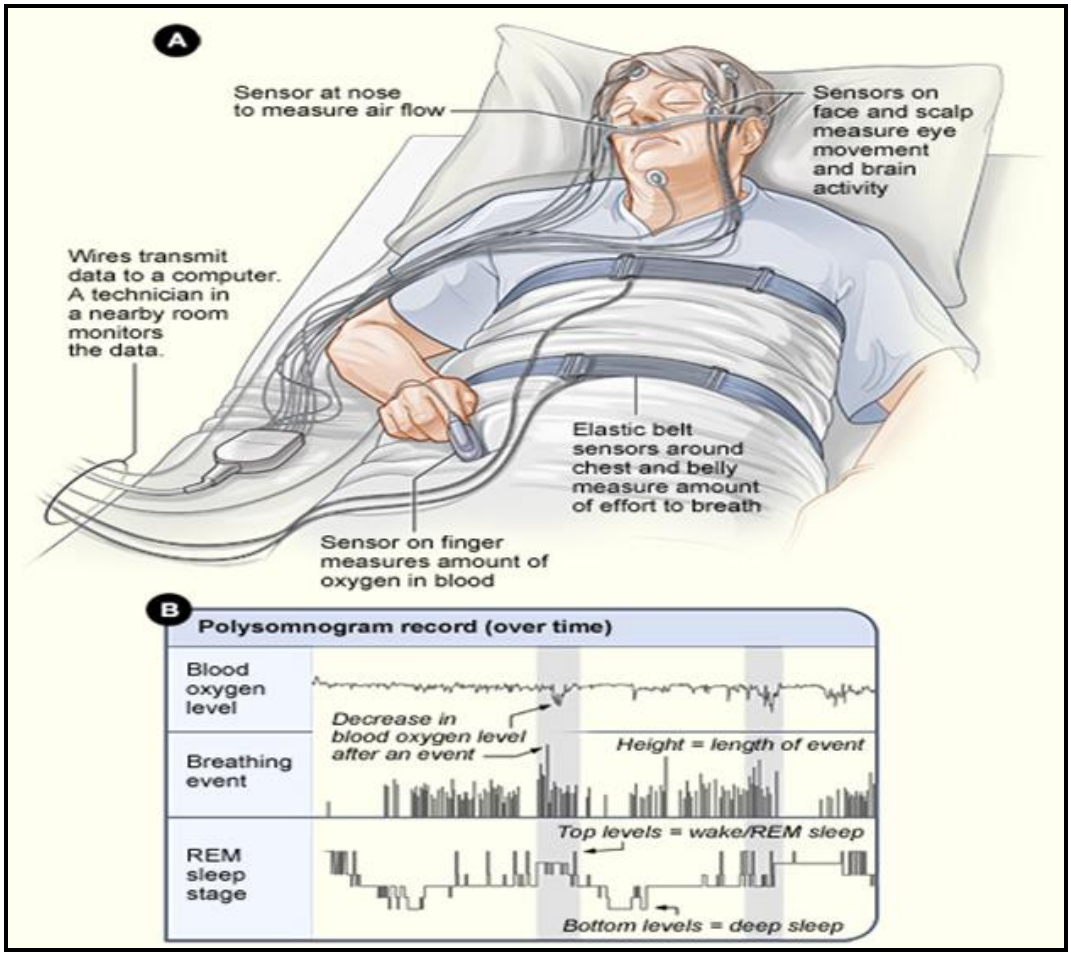
2) Total sleep time(TST)-" actual sleep REM +NREM"

3) Sleep period time(SPT)- " sleep onset to final awakwning"

4) Sleep efficiency (SE)-" percentage of TST in total time in bed". SE>85%is normal

5) Sleep latency (SL) –"time elapsed between lights out to the first epoch of sleep (usually stage 1)".



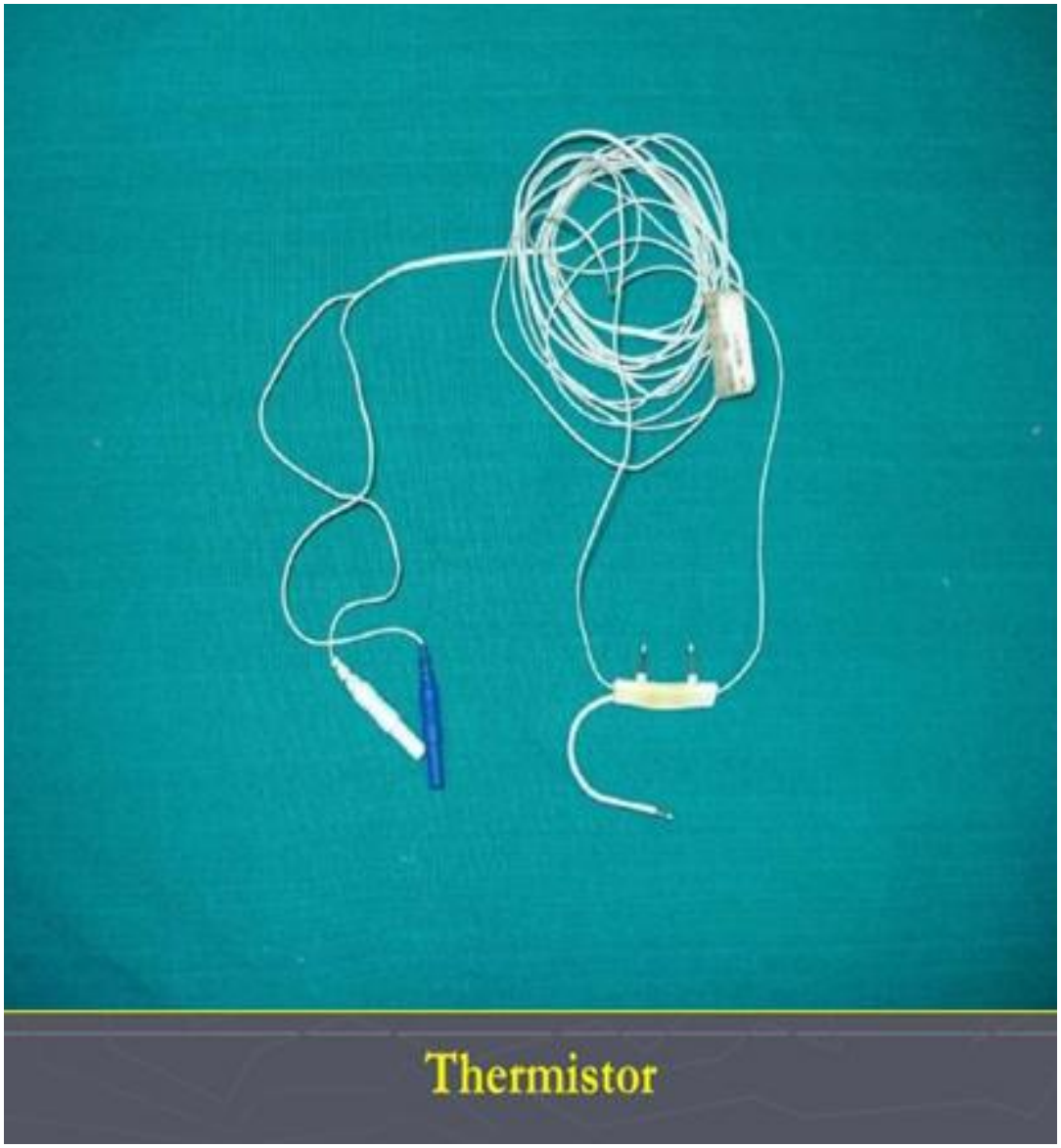

A
Sensor at nose to measure air flow
Sensors on face and scalp measure eye movement and brain activity
Wires transmit data to a computer. A technician in a nearby room monitors the data.
Elastic belt sensors around chest and belly measure amount of effort to breath
Sensor on finger measures amount of oxygen in blood
B
Polysomnogram record (over time)
Blood oxygen level
Breathing event
REM sleep stage
Decrease in blood oxygen level after an event
Height = length of event
Top levels = wake/REM sleep
Bottom levels = deep sleep

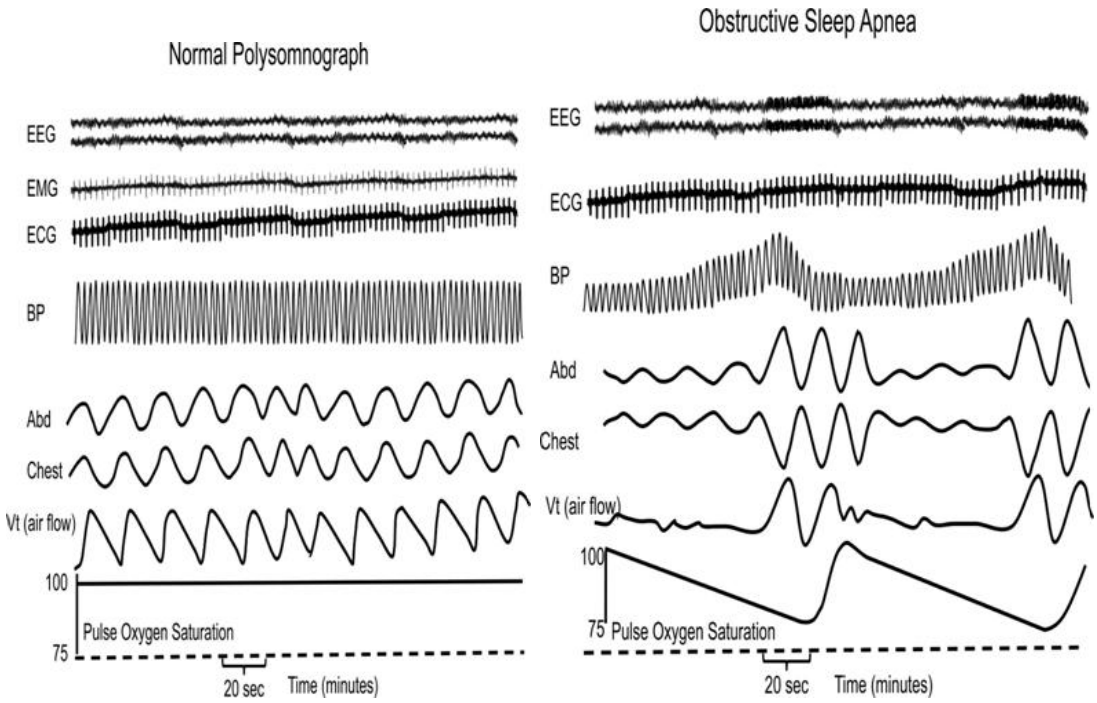

Transducer for thoracoabdominal movement

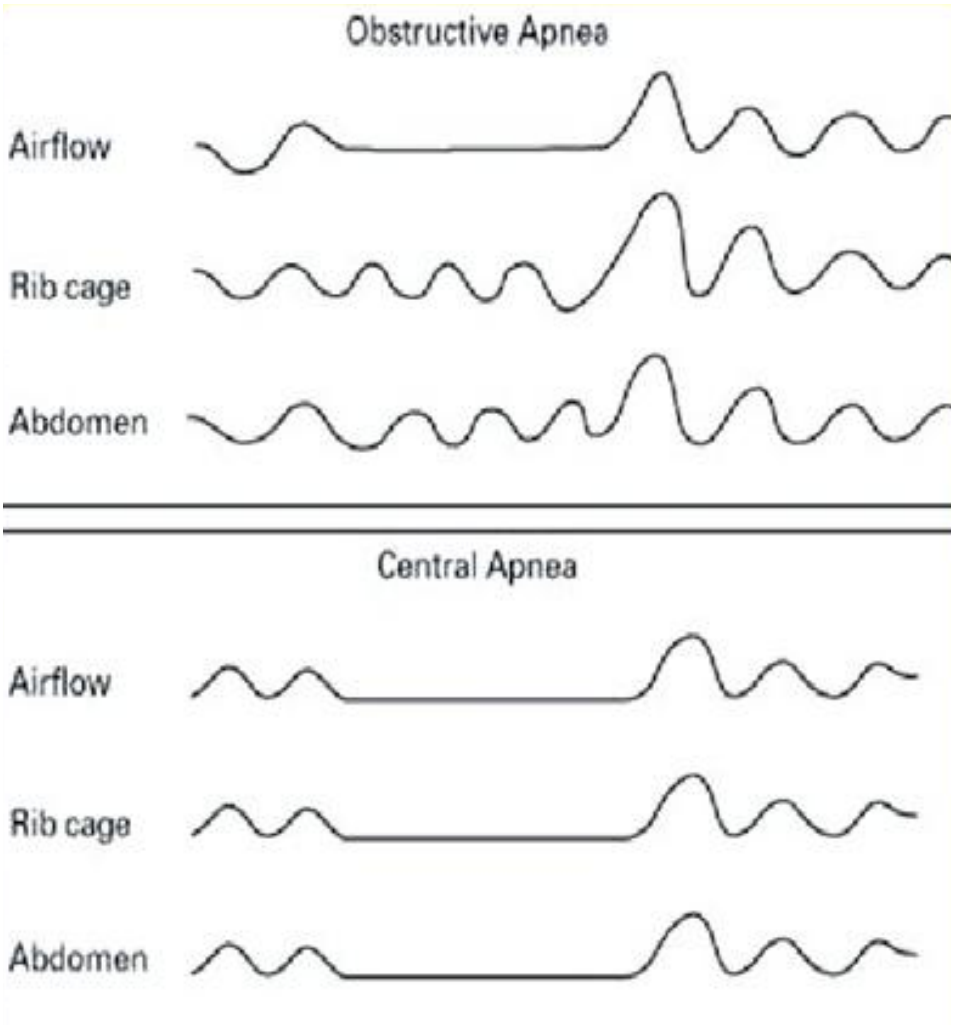
PLETHYSMOGRPHY

1) Impedence plethysmography- measures the elastic recoil during respiration into electric impulses

2) Magnetometry- measures magnetic changes between the two sides of chest




Normal Polysomnograph
EEG
EMG
ECG
BP
Abd
Chest
Vt (air flow)
100
Pulse Oxygen Saturation
75
20 sec
Time (minutes)
Obstructive Sleep Apnea
EEG
ECG
BP
Abd
Chest
Vt (air flow)
100
75 Pulse Oxygen Saturation
20 sec
Time (minutes)


Obstructive Apnea
Airflow
Rib cage
Abdomen
Central Apnea
Airflow
Rib cage
Abdomen

FLUROSCOPY

Fluoroscopy is a readily available technique to assess dynamic airway anatomy and sites of obstruction in OSA patients. Somnofluoroscopy combines fluoroscopy with polysomnography and radiologically evaluate thesites of obstruction during episodes of apnea and hypopnea. Advantages of fluoroscopy include direct observation of obstructive sites during episodes of apnea, and availability of fluoroscopy in most hospitals.

Drawbacks include high radiation dose, superimposition of structures,and the possible need for sedation to attain sleep during the procedure. Newe rdigital fluoroscopy systems require less radiation exposure and shorter examination times.

MANOMETRY

Manometry techniques use catheters in the upper airway to measure pressure at various sites in the upper airway. Patients who do not have frank apneas or hypopneas, but had symptoms of OSA, short alpha EEG arousals during sleep, and abnormal increases in upper airway resistance are measured using manometry during sleep have categorized into upper airway resistance syndrome. CPAP has been shown to resolve the symptoms associated with upper airway resistance syndrome. Both awake and asleep manometry measurements have been performed to identify areas at risk for collapse. In awake patients, externally provided negative pressure or patient provided negative pressure (MM) have been used to collapse the airway during evaluation. A sleep manometry has the significant advantages of assessing airway collapsibility without externally induced pressures, and of removing

the confounding effect of airway muscular tone. Manometry studies are difficult to perform in that they require precise placement of an invasive probe transnasally, which is poorly tolerated by many patients. Typically, a sleep manometry is performed in concurrent with sleep studies, and allows for measurement of intra thoracic pressure and respiratory drive to assist with identification of central apnea.

TREATMENT OPTIONS

Treatment options for OSA fall under three categories :

1) Behavioural Modification

2) Devices that can be worn

3) Surgery

BEHAVIOURAL MODIFICATION

WEIGHT REDUCTION

Weight reduction leads to improvement in lung volumes thereby reducing pharyngeal resistance and improves nocturnal hemoglobin saturation.

SLEEP POSTURE THERAPY

The patients are asked to sleep in lateral positions or in prone position.

DEVICES THAT CAN BE WORN

Positive airway pressure:

Most effective treatment in moderate to severe grades of OSA in tolerant patients. It is available in many forms such as CPAP, biPAP, automatically titrating PAP and demand PAP.(8) Each delivers positive

pressure through a device worn on the face, and serves as an internal pneumatic splint for the airway. CPAP, the most commonly used form of PAP, typically uses between 5 and 15 cm of water pressure to maintain airway patency.(8)

Oral appliances

Mandibular repositioning devices advance the mandible anteriorly, which brings forward the tongue and other muscles of the oropharynx and hypopharynx.The position of the palate is also changed with the mandibular repositioning device through action of the palatoglossus muscle. It is used for patients with simple snoring and mild apnea. Patients with pre-existing disorders of the tempero mandibular joint and edentulous patients are not considered to be good candidates for this device. As with PAP, no training of the airway occurs, therefore nightly use is necessary for treatment effect.(9)

Tongue - Retaining Device (TRD)

" It increases pharyngeal patency by pulling the superior aspect of the tongue forward, away from the posterior wall of the pharynx" .(9)

SURGICAL MANAGEMENT OF OSA

UTILITY OF SURGERY IN OSA

1) "By passing the obstructive area by tracheostomy

2) eliminating the obstructive lesion in order to prevent soft palate collapse in the upper airway during an apneic episode"

Indications include

1) "Morbidly obese patients unable to tolerate PAP

2) Morbidly obese patients with Pickwickian syndrome who require upper airway bypass and nocturnal ventilation

3) Patients Who are unable to tolerate PAP

4) Other Treatment options have failed".

Bariatric surgery is considered when

1) BMI > 40 or

2) BMI > 35 with significant co-morbidities.

UPPER AIRWAY SURGERY IN OSA PATIENTS

INDICATIONS

1) AHI >20 events / hour.

2) Oxygen desaturation nadir'>90%

3) 'Esophageal pressure more negative' '> -10cm H2O'

4) 'Cardiovascular derangements' (arryhtmias, elevated blood pressure)

5) 'Neurobehavioural symptoms' (excessive daytime sleepiness)

6) Failure of medical management

7) 'Anatomical sites of obstruction' (nose, tongue, softpalate)(10)

CONTRAINDICATIONS

1) Severe pulmonary disease

2) Morbid obesity

3) Unstable cardiovascular disease

4) Alcohol or drug abuse

5) Psychiatric instability

"POWELL RILEY TWO PHASE SURGICAL PROTOCOL'' "PHASE 1'':

- "nasal surgery' (septoplasty , turbinate reduction , nasal valve grafting)
- Uvulopalatopharyngoplasty
- 'Tonsillectomy'
- 'Mandibular osteotomy with genioglossus advancement'
- 'Hyoid myotomy and suspension'
- 'Temperature controlled radiofrequency' (TCRF)- turbinates , palate, tongue base."(11)

"PHASE 2''

- 'Maxillomandibular Advanced Osteotomy'
- 'Temperature controlled radiofrequency (TCRF)'- tongue base

UTILITY OF '' POWELL RILEY SURGICAL PROTOCOL''

- ❖ Offers a tailor made approach for each patient
- ❖ Procedures are focused towards specific pathology
- ❖ Risk of over operating is reduced. (11)
- ❖ Limited pain and reduced time for regaining well being
- ❖ Acceptable for many
- ❖ Improved cure rates for phase 2 surgery

SURGICAL TREATMENT OF OSA

Depending upon the level of obstruction various surgical options are available(12)

Palatal	"Z-pharyngoplasty, injection snoreplasty, Modified UPPP with extended Uvulopalatal flap, palatal implants"
Lateral pharyngeal wall	"Lateral pharyngoplasty,Expansion sphincter pharyngoplasty "
Tongue Base	"Laser midline glossectomy and lingualplasty, tongue suspension, lingual tonsillectomy, RFVTR, Hypoglossal nerve stimulation"
Epiglottis	"Endoscopic epiglottectomy"
Trachea	"Temporary tracheostomy"
CRANIOFACIAL ABNORMALITIES	"MMP,HMS"

AIMS OF THE STUDY

1) To identify the level of obstruction in obstructive sleep apnea patients

2) To compare the level of obstruction seen in normal sleep cine mri with that of drug induced sleep endoscopy

MATERIALS AND METHODS

STUDY PLACE

Rajiv Gandhi Government General Hospital, Chennai – 600003

COLLABORATING DEPARTMENT

Upgraded Institute of Otorhinolaryngology and Barnard institute of radiology.

STUDY DESIGN

Retrospective and Prospective study

STUDY PERIOD

November 2013 To September 2015

STUDY POPULATION

All patients with snoring and OSA who reported to the upgraded Institute of Otorhinolaryngology of Madras Medical College ,during the study period with the fulfillment of inclusion criteria .

INCLUSION CRITERIA

1) Age between 20 and 50 yrs

2) Both sexes (Male and Female)

3) BMI <40

4) Neck circumference >17 inches for men and >16 inches for women

5) Epsworth Sleepiness Scale >10

6) AHI>5

EXCLUSION CRITERIA

1) Age below 20yrs and above 50yrs

2) Hypothroidism and other metabolic disorders

3) BMI>40

4) Associated craniofacial abnormalities

INVESTIGATIONS

1) Thyroid Function Test

2) Polysomnography

3) FiberopticNasopharyngoscopy

4) DISE (Drug induced sleep endoscopy)

5) Cephalometry

6) Dynamic MRI

ETHICAL COMMITTEE APPROVAL

Institutional Ethical Committee, Government General Hospital, Madras Medical College, Chennai reviewed the experimental design and protocol as well as the letter of information and consent form. Full approval of the board was granted. All patients were given information outlining the experimental protocol and all patients signed a consent form prior to entering the study.

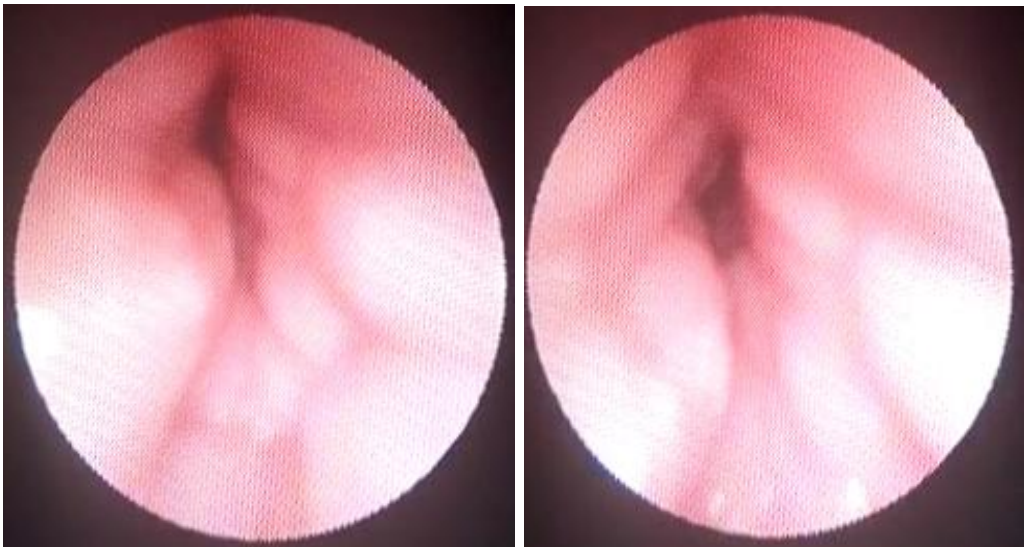
METHODOLOGY

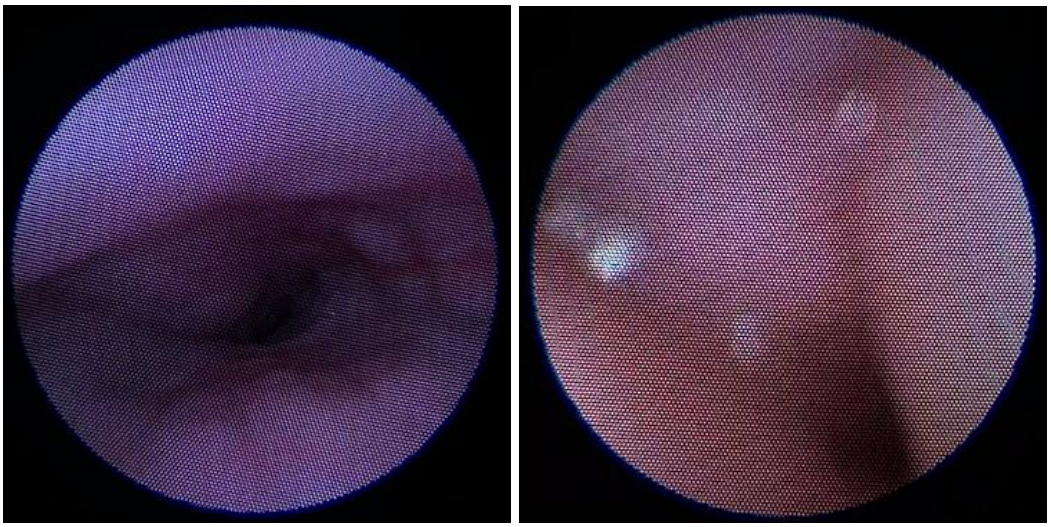
This is a prospective study conducted in our institution from November 2013 to September 2015.All patients who attend our op with the complaints of snoring, frequent awakening at night ,excessive day time sleepiness ,hallucinations. choking in sleep are further evaluated.All the patients undergo clinical examination followed by blood investigations especially thyroid function test and BMI evaluation.These patients were then subjected to overnight polysomnography.Those patients with AHI>5 and those who fulfil the inclusion criteria were then subjected to dynamic MRI and DISE (drug induced sleep endoscopy).

DRUG INDUCED SLEEP ENDOSCOPY

Between November 2013 to September 2015 Patients who fulfilled the inclusion criteria were subjected to DISE identify the level of obstruction.The level of obstruction in OSAS is usually at 4 levels namely velum, oropharynx, tonguebase and epiglottis.identifying the level of obstruction is important to decide the treatment plan for the patient (21,23,25,27).DISE provides valuable information such as structure, pattern and degree of collapsibility .It also helps us to anticipate difficult intubation/extubation .After obtaining anesthetic fitness patient is shifted to operating room .Patient is made to lie supine in the operating table and iv line is secured and RL infusion is started .Patient is pre oxygenated with 100% o2.LMA and ET tubes kept ready if in any case saturation levels falls .Inj.propofol is gradually given iv for the patient to snore.It is started at a dose of 0.5mg/kg(18,19,20) and can be increased upto 1 mg/kg .Usually the total dose is <50mg.after the patient starts snoring surgeon who is standing at the head end of the patient

introduces flexible nasopharyngoscope through patients nostril and visualizes the upper airway to identify the level of obstruction and degree of collapasability.instead of iv propofol ,(22,24) iv dexmedetomidine in dose of 0.5-1micrograms/kg can also be used to sedate the patient.(13,14,15)



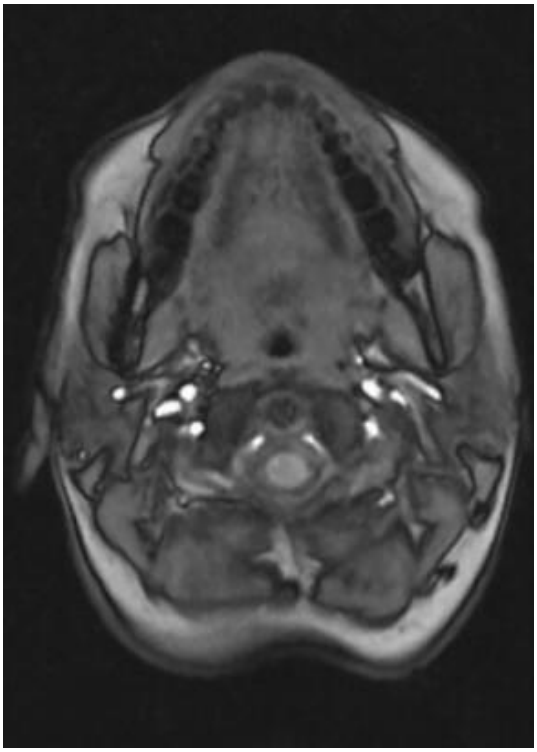


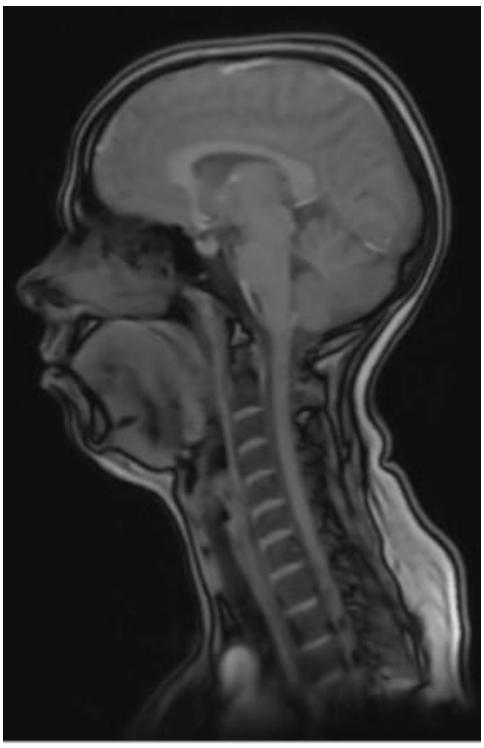
Grade	Obstruction
1	Simple palatal flutter
2	Single level palatal obstruction
3	Palatal level with intermittent oropharyngeal involvement
4	Sustained multisegmental obstruction
5	BOT obstruction
6	Isolated epiglottic involvement

DYNAMIC MRI

Between November 2013 to September 2015 35 patients who were diagnosed as osas fulfilling the inclusion criteria were subjected to sleep MRI study after obtaining proper consent.Dynamic MRI was taken in 3 positions (i.e) awake,muller manuvere ,normal sleep.Patients were examined at barnard institute of radiology at rajiv Gandhi govt. general hospital under continuous supervision by a radiologist .Patients were refrained from sleep 20 hrs before the MRI studies and were not allowed to ingest alcohol or sedatives during the day of the procedure .studies were carried out at night time .Axial and saggital images of the upper airway at the level of velum,oropharynx, tongue base,hypopharynx obtained in all 3 positions .A-P and Lateral diameters of upper airway measured at the level of velum, oropharynx and hypopharynx.50% reduction in upper airway dimension from awake position was considered as obstruction .Continuous spo2 monitoring was done during the procedure .Respiratory motion was monitored by a magnetic resonance linked cuff placed at the rib cage level .wakefullness is documented by

patient’s response to the radiologist, During muller manuevere patient is asked to “attempt a forceful inspiration against closed nose and mouth” which is opposite to valsalva manuevere and upper airway dimensions measured .sleepfullness is documented by snoring heared by technician and fall in spo2 levels by atleast 4%.(30,31)





STATISTICAL ANALYSIS AND RESULTS

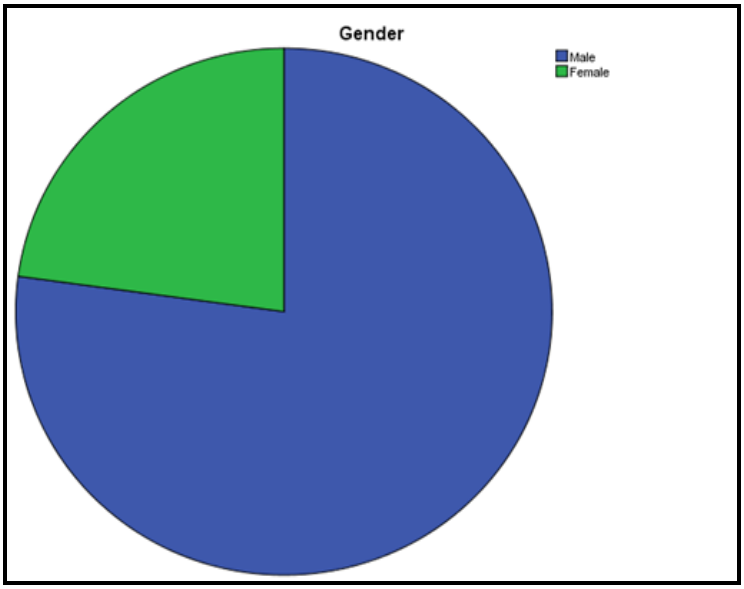
N	Valid	35
	Missing	0

GENDER

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Male	27	77.1	77.1	77.1
	Female	8	22.9	22.9	100.0
	Total	35	100.0	100.0	

77.1% Population in the study were male patients (27/35)

22.9% population in the study were females (8/35).



AHI

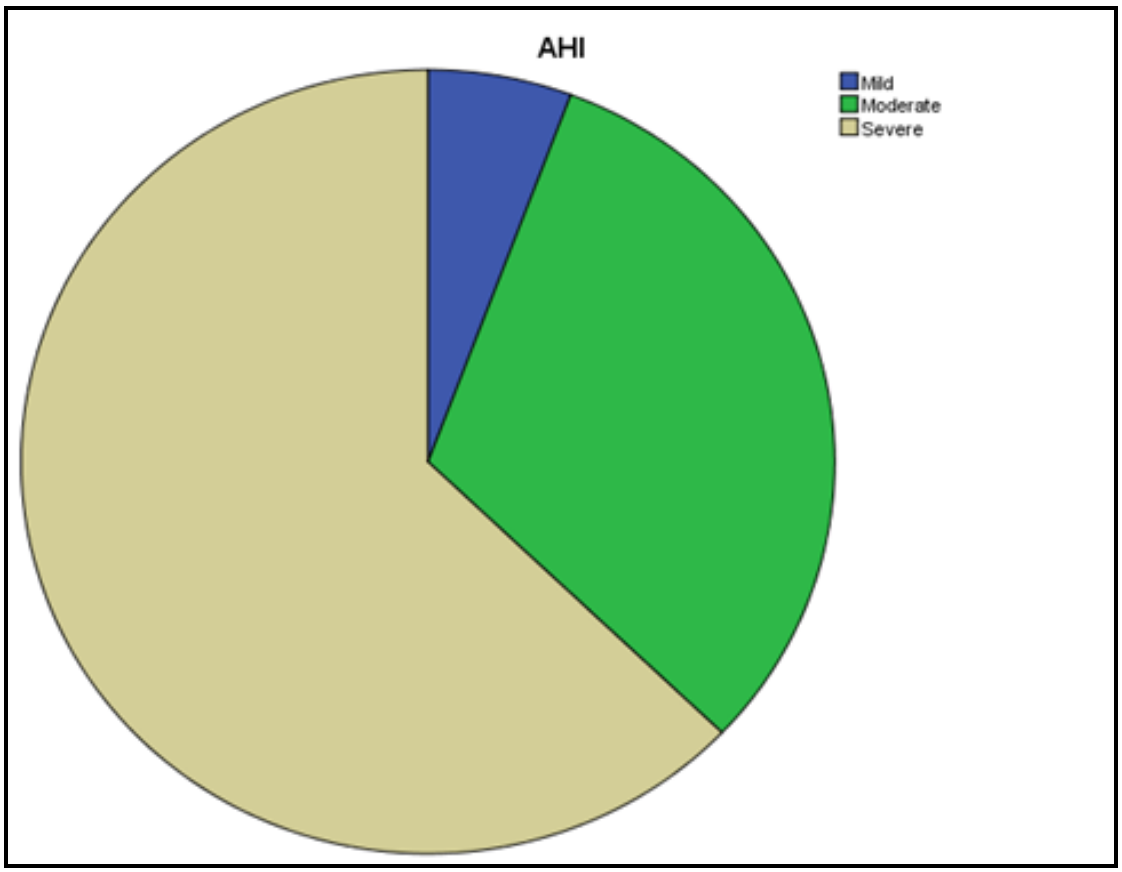
N	Valid	35
	Missing	0

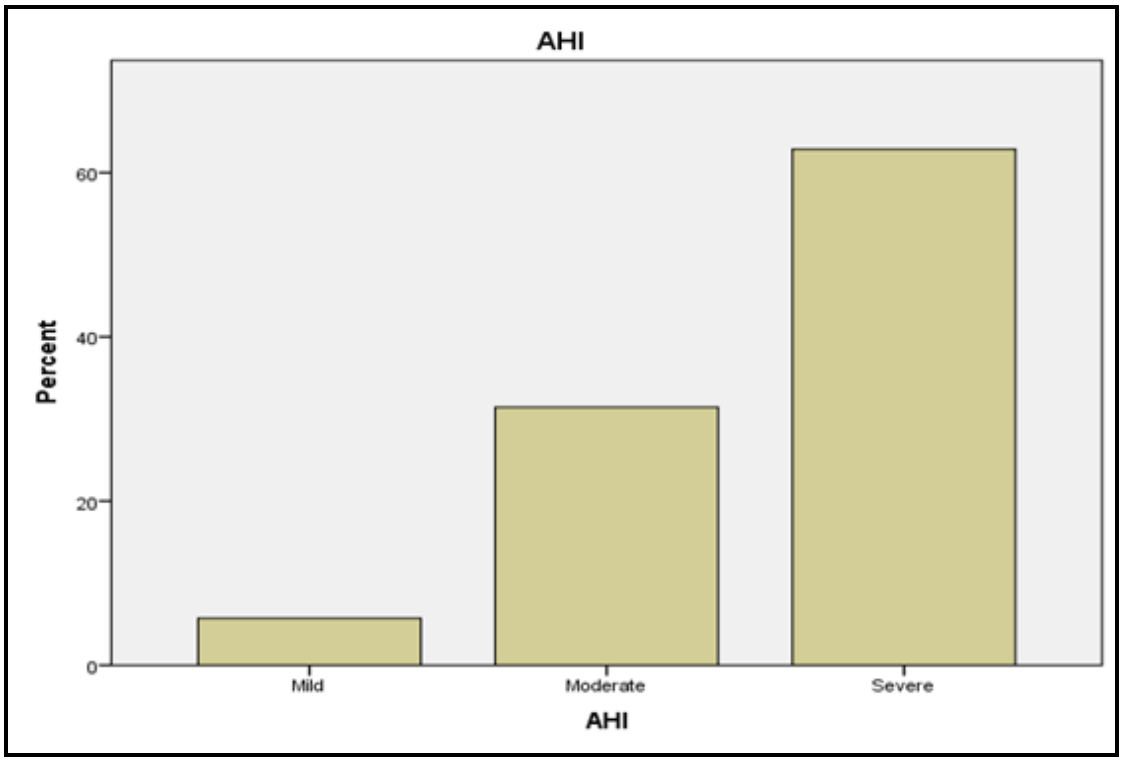
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Mild	2	5.7	5.7	5.7
	Moderate	11	31.4	31.4	37.1
	Severe	22	62.9	62.9	100.0
	Total	35	100.0	100.0	

In the study 5.7% were mild OSA patients (2/35).

31.4% were moderate OSA patients (11/35).

62.9% were severe OSA patients (22/35).


AHI
Mild
Moderate
Severe


AHI
Percent
60
40
20
0
Mild
Moderate
Severe
AHI

MEANSPO2

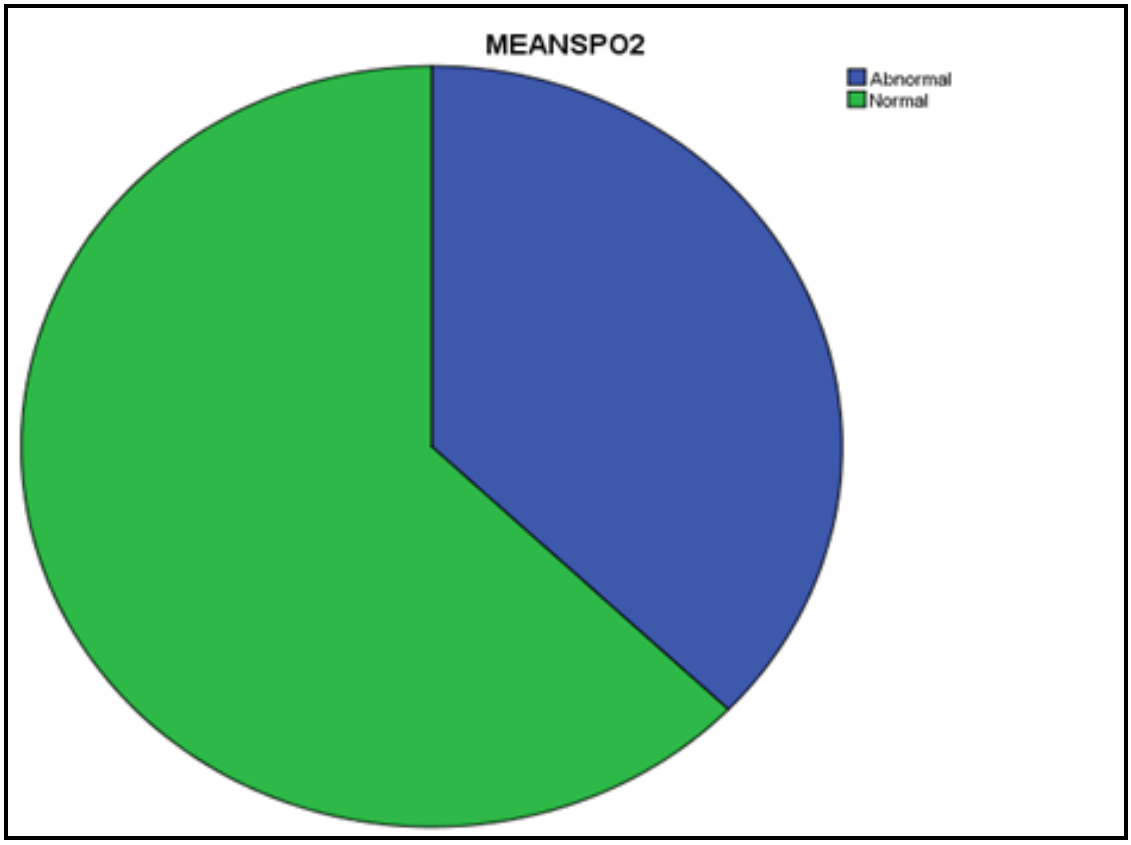
N	Valid	35
	Missing	0

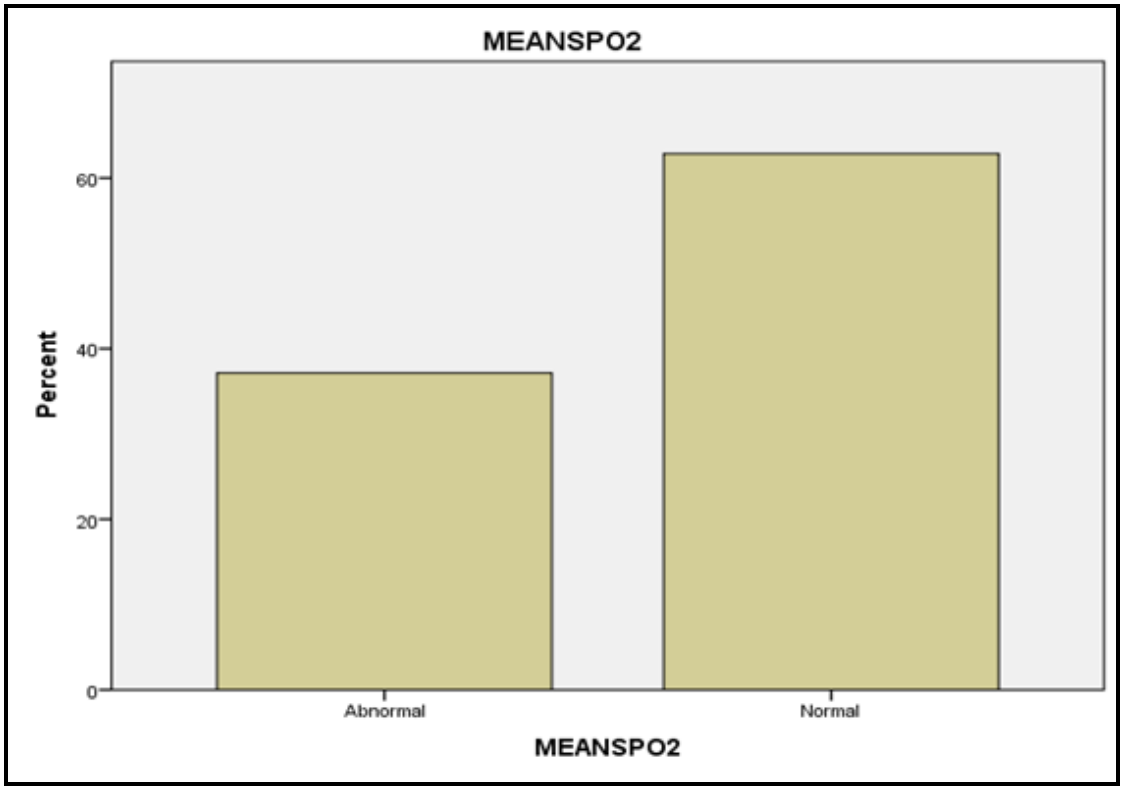
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Abnormal	13	37.1	37.1	37.1
	Normal	22	62.9	62.9	100.0
	Total	35	100.0	100.0	

Mean spo2<90% taken as abnormal.

37.1% population had mean SPO2<90%.

62.9% population had mean SPO2>90%.


MEANSPO2
Abnormal
Normal


MEANSPO2
Percent
60
40
20
0
Abnormal
Normal
MEANSPO2

LENGTH OF SOFT PALATE

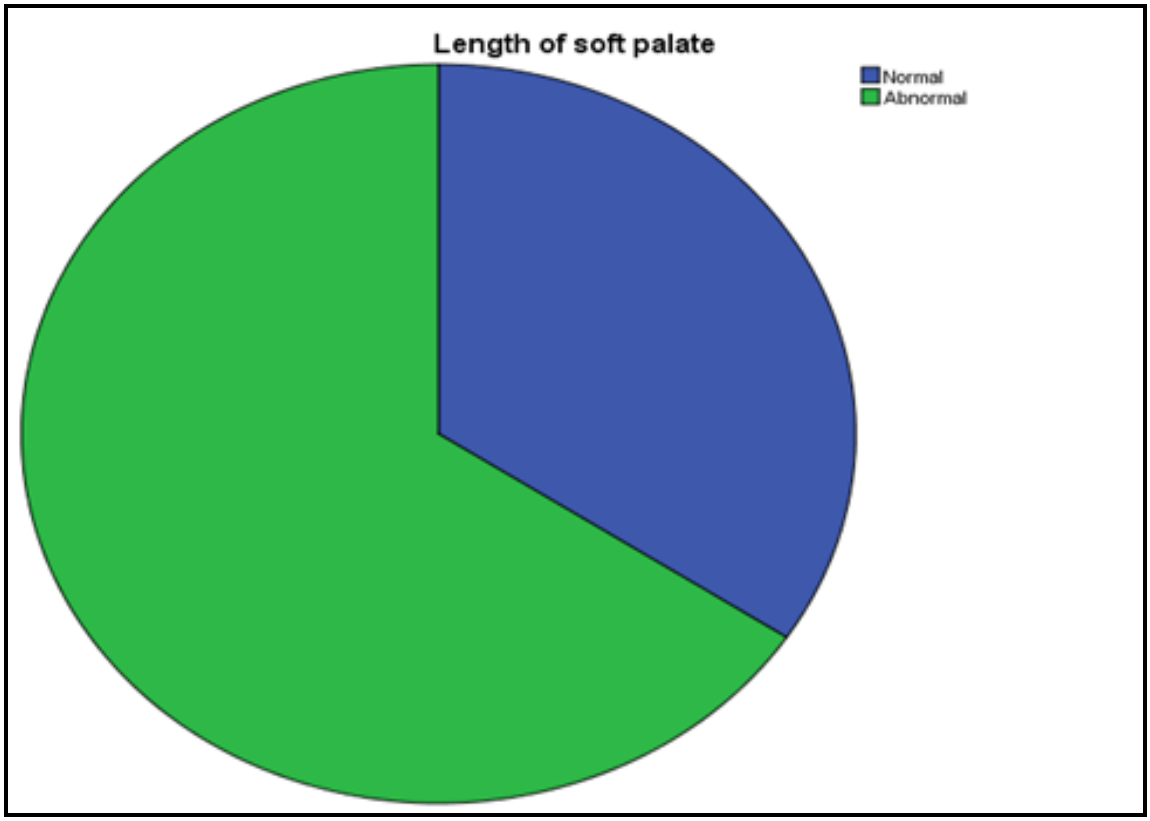
N	Valid	35
	Missing	0

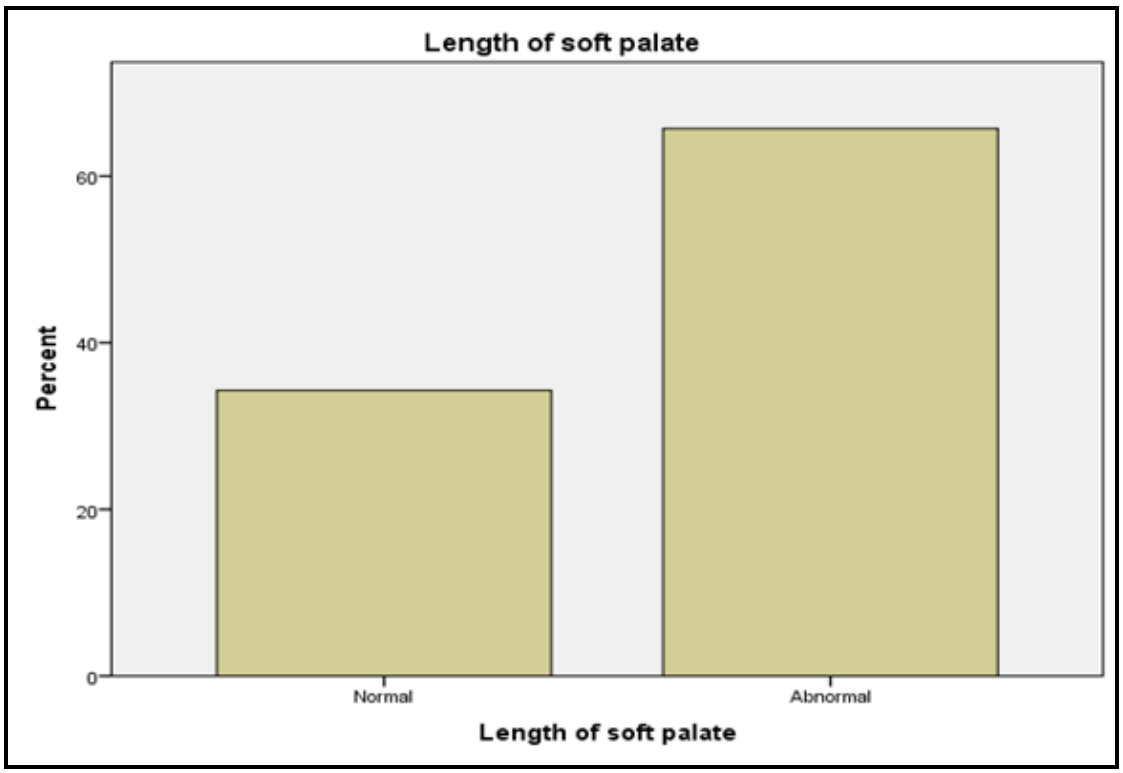
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	12	34.3	34.3	34.3
	Abnormal	23	65.7	65.7	100.0
	Total	35	100.0	100.0	

Length Of Soft Palate <3.5cm was taken as normal Length of soft palate >3.5 cm was taken as abnormal.

34.3%population in the study had normal soft palate length(12/35).

65.7% population in the study had abnormal soft palate length(23/35).


Length of soft palate
Normal
Abnormal


Length of soft palate
Percent
60
40
20
0
Normal
Abnormal
Length of soft palate

THICKNESS OF SOFT PALATE

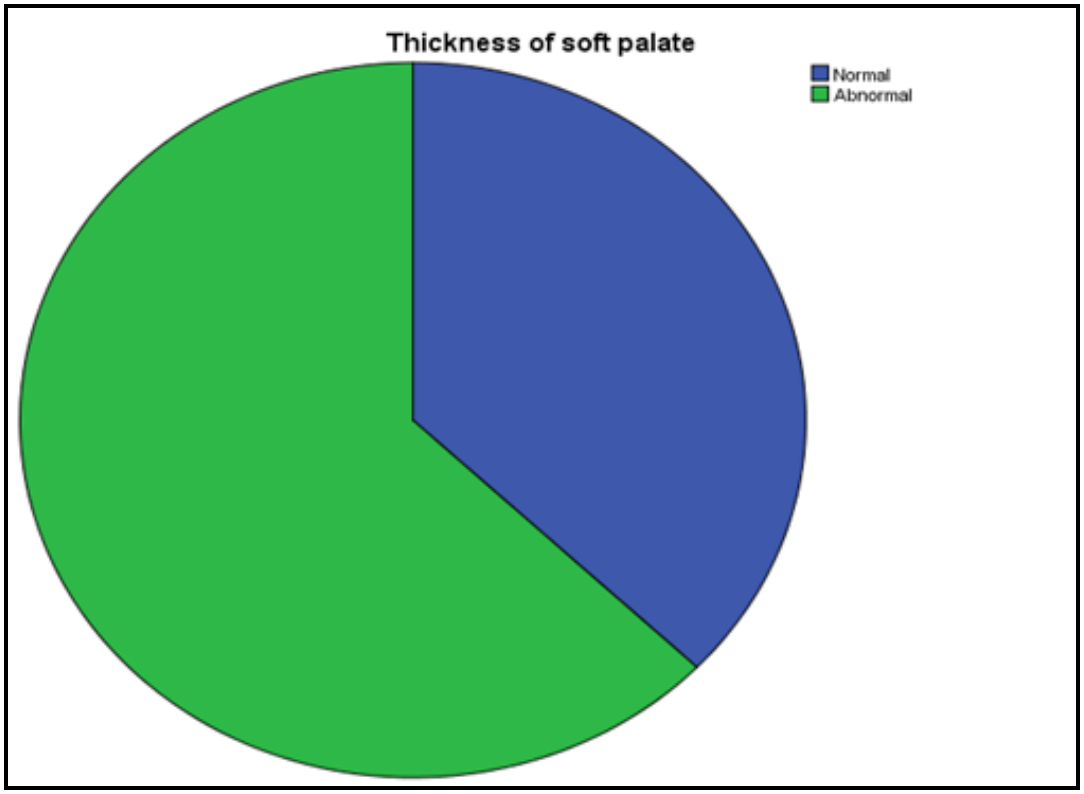
N	Valid	35
	Missing	0

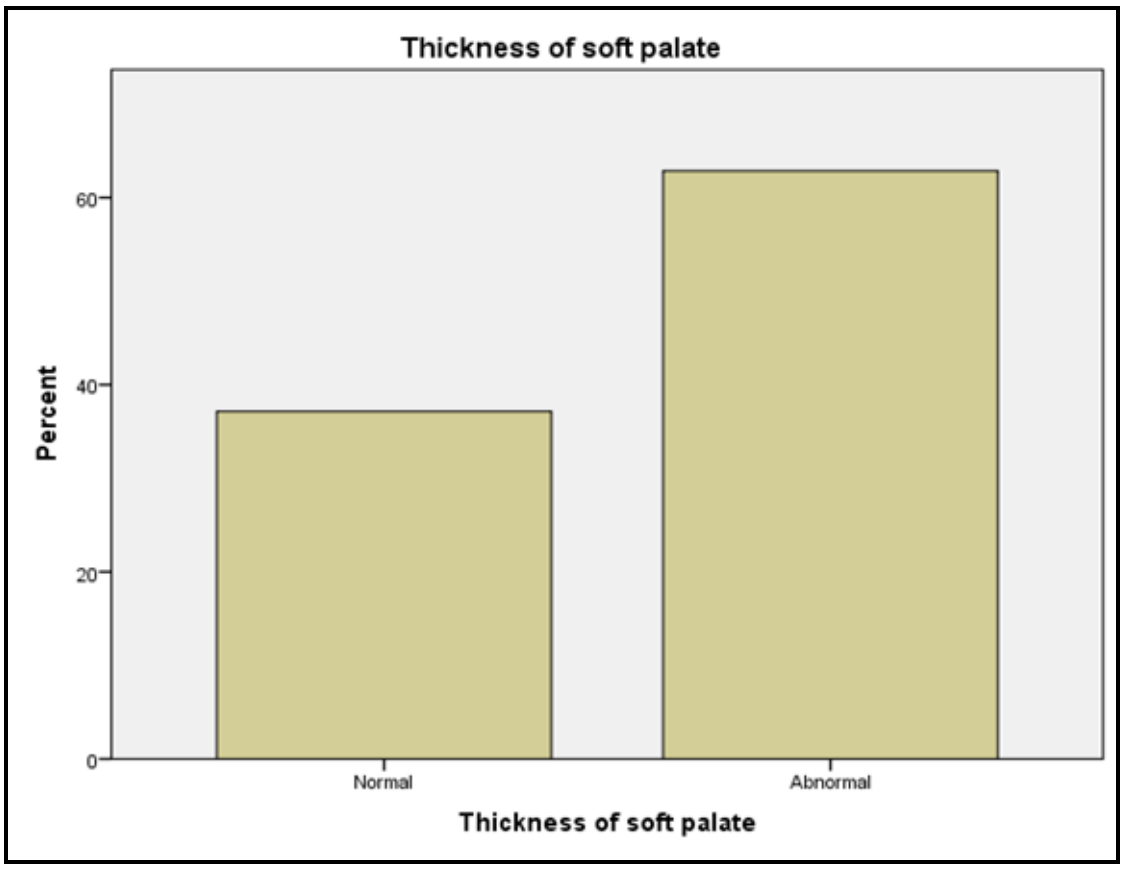
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	13	37.1	37.1	37.1
	Abnormal	22	62.9	62.9	100.0
	Total	35	100.0	100.0	

Thickness of soft palate <10mm was taken as normal

Thickness of soft palate>10 mm was taken as abnormal.

62.9% of the study population had abnormal soft palate thickness.


Thickness of soft palate
Normal
Abnormal


Thickness of soft palate
Percent
60
40
20
0
Normal
Abnormal
Thickness of soft palate

AGE

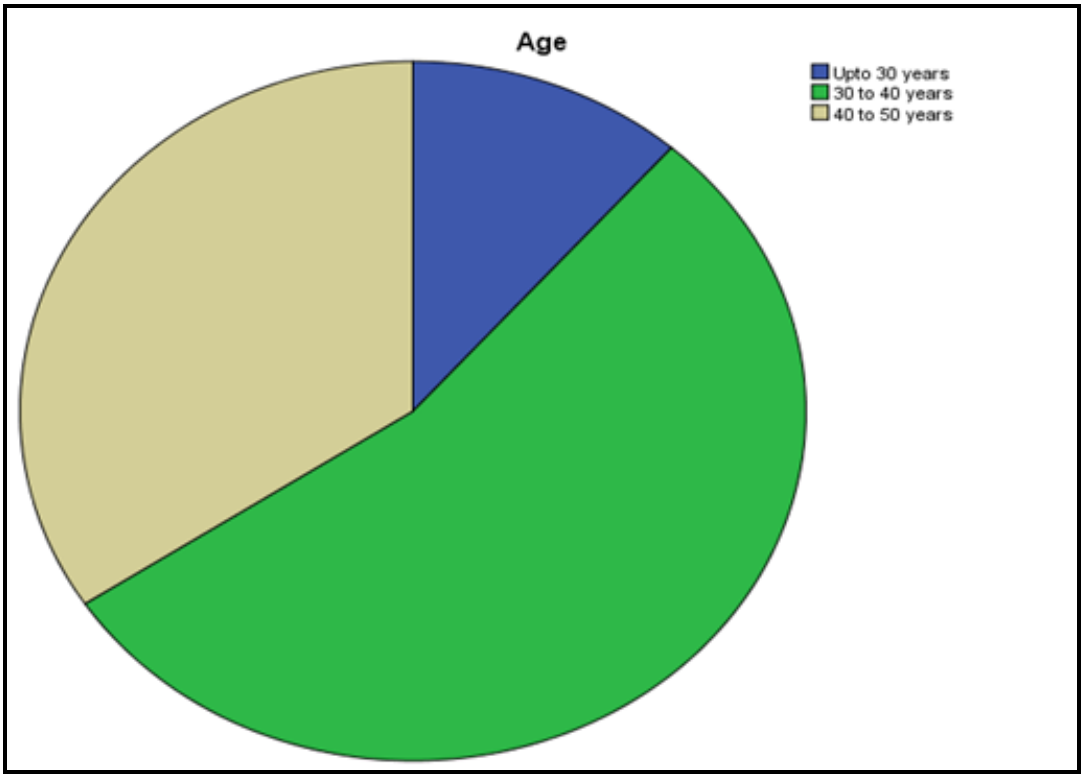
N	Valid	35
	Missing	0

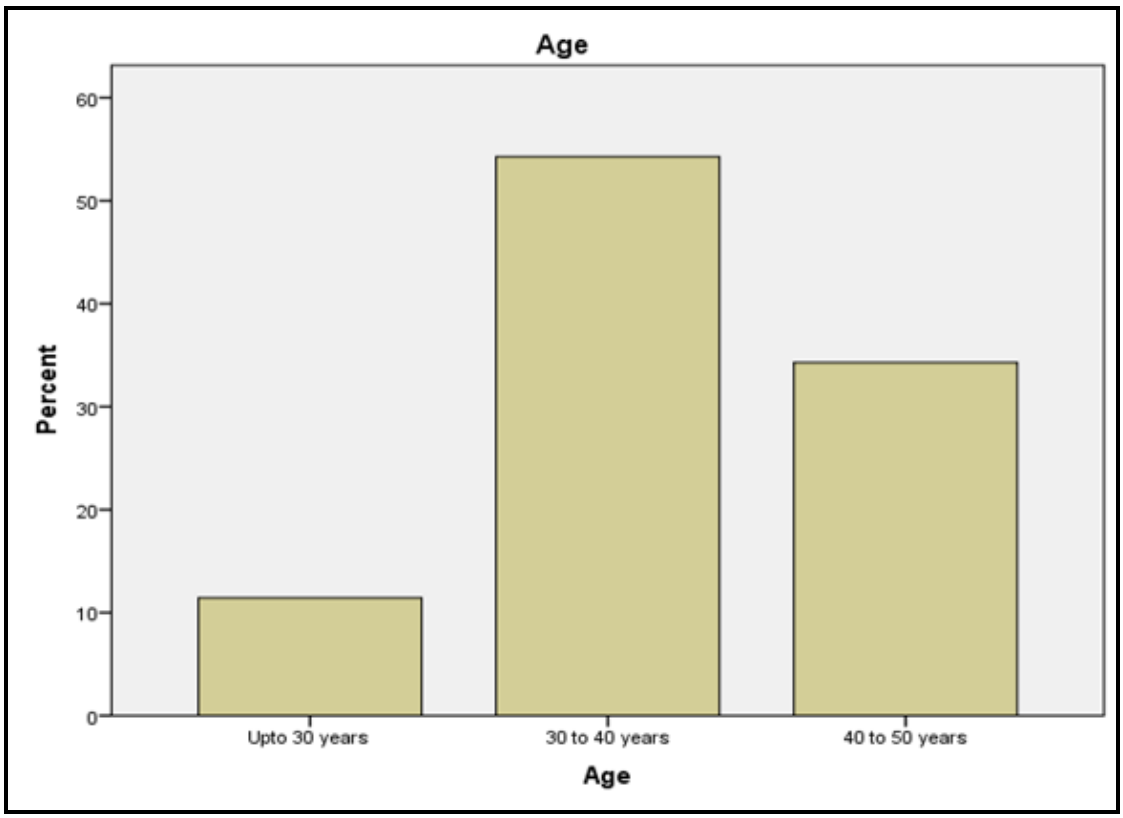
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Upto 30 years	4	11.4	11.4	11.4
	30 to 40 years	19	54.3	54.3	65.7
	40 to 50 years	12	34.3	34.3	100.0
	Total	35	100.0	100.0	

11.4% of the study population were <30 years

54.3% of the study population were 30-40years

34.3% of the study population were 40-50 years


Age
Upto 30 years
30 to 40 years
40 to 50 years


Age
Percent
60
50
40
30
20
10
0
Upto 30 years
30 to 40 years
40 to 50 years
Age

DISE GRADING

N	Valid	35
	Missing	0

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Palate obst	12	34.3	34.3	34.3
	Palate and Oro	9	25.7	25.7	60.0
	Multi segment	2	5.7	5.7	65.7
	Tongue base	4	11.4	11.4	77.1
	Epiglottis	8	22.9	22.9	100.0
	Total	35	100.0	100.0	

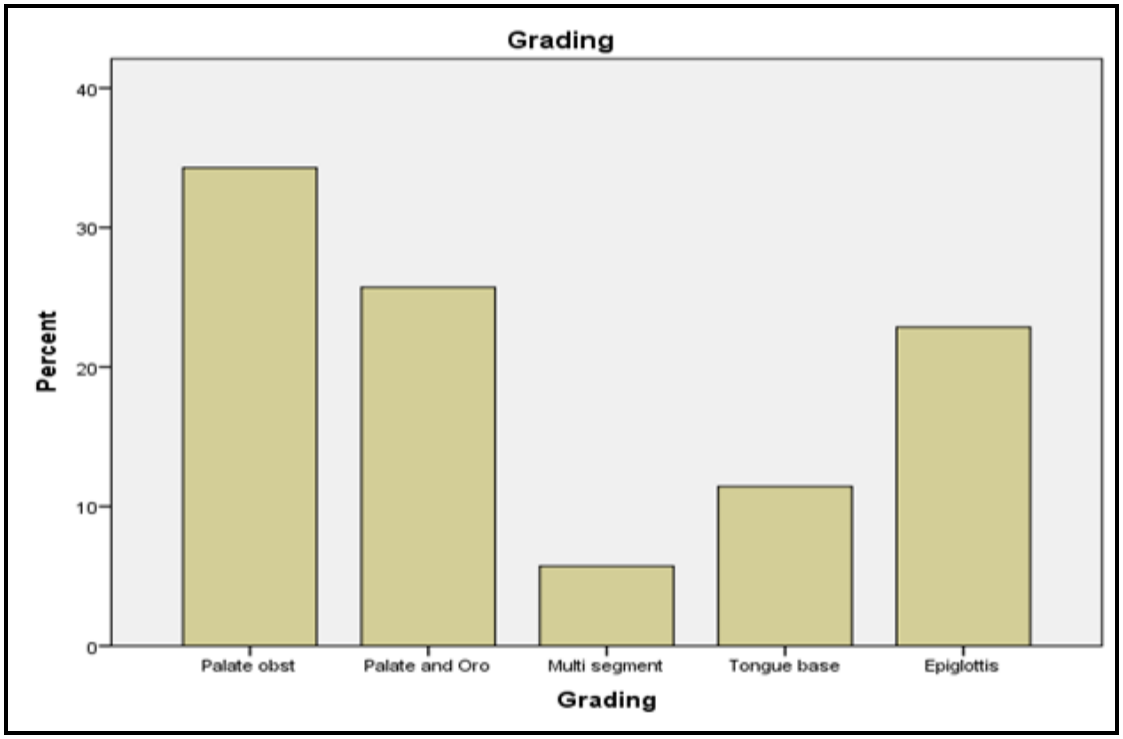
34.3% OF Study population belonged to grade 2 in DISE

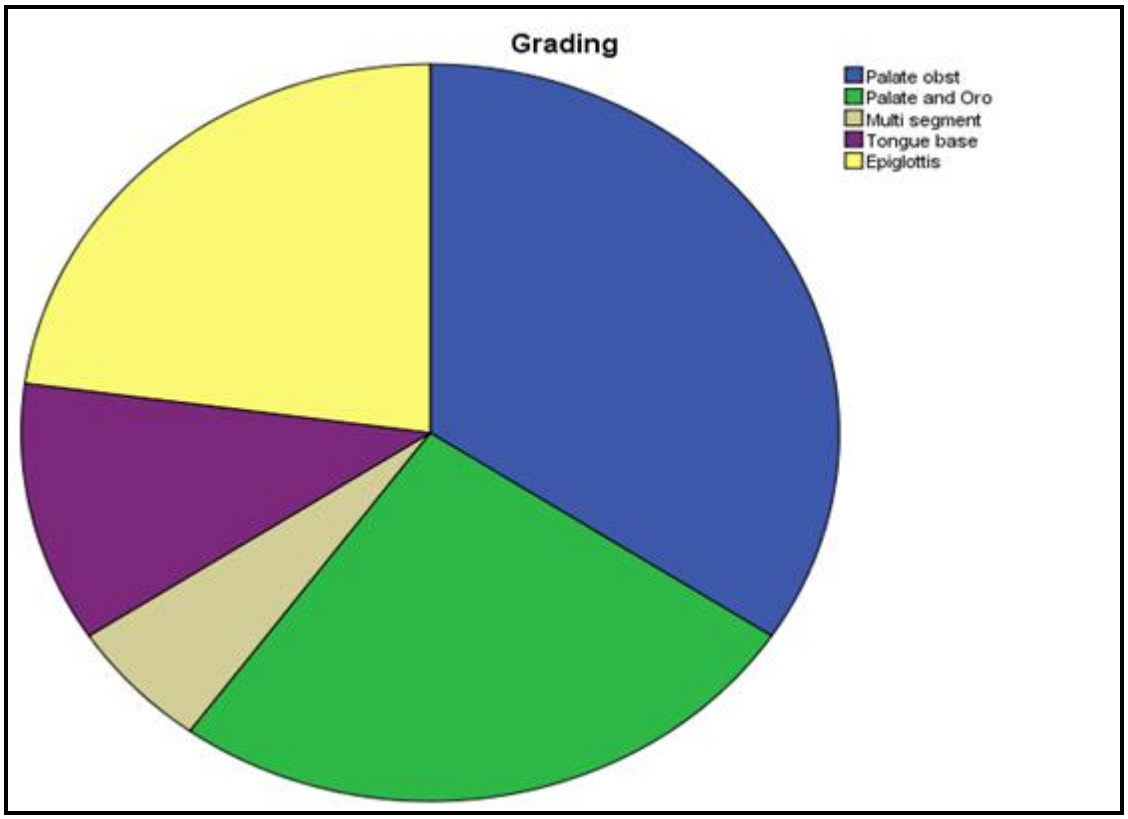
25.7% of study population belonged to grade 3 in DISE

5.7% of study population belonged to grade 4 in DISE

11.4% of study population belonged to grade 5 in DISE

22.9% of study population belonged to grade 6 in DISE


Grading
Percent
40
30
20
10
0
Palate obst
Palate and Oro
Multi segment
Tongue base
Epiglottis
Grading


Grading
Palate obst
Palate and Oro
Multi segment
Tongue base
Epiglottis

LEVEL OF OBSTRUCTION IN MRI

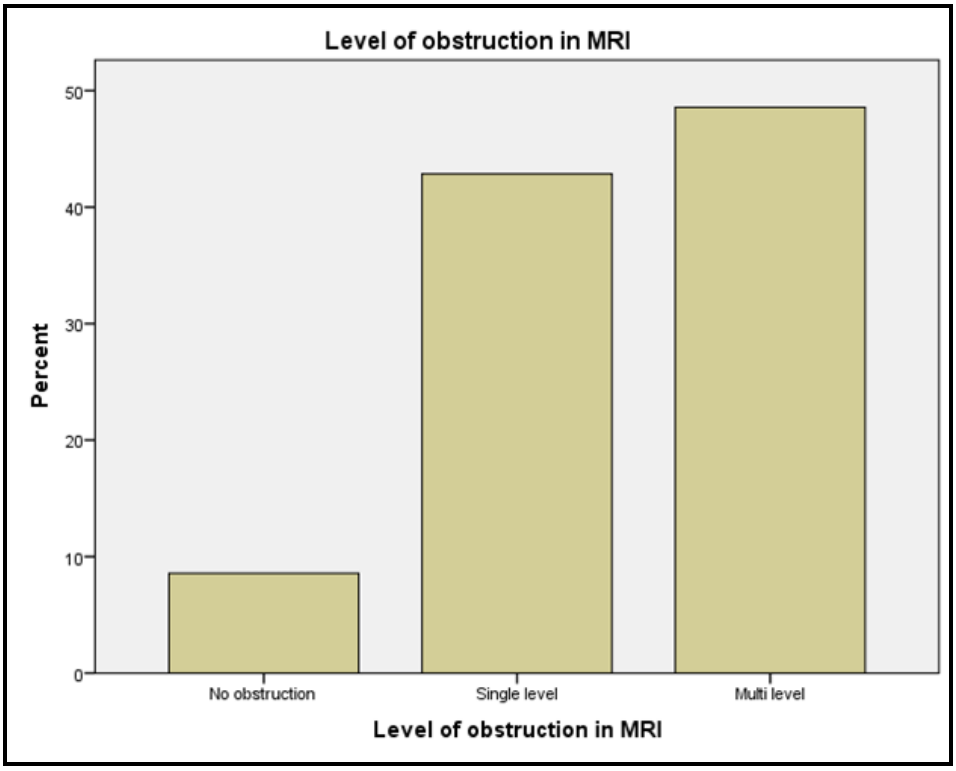
N	Valid	35
	Missing	0

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No obstruction	3	8.6	8.6	8.6
	Single level	15	42.9	42.9	51.4
	Multi level	17	48.6	48.6	100.0
	Total	35	100.0	100.0	

In the above table 8.6%patients had no obstruction in dynamic MRI.

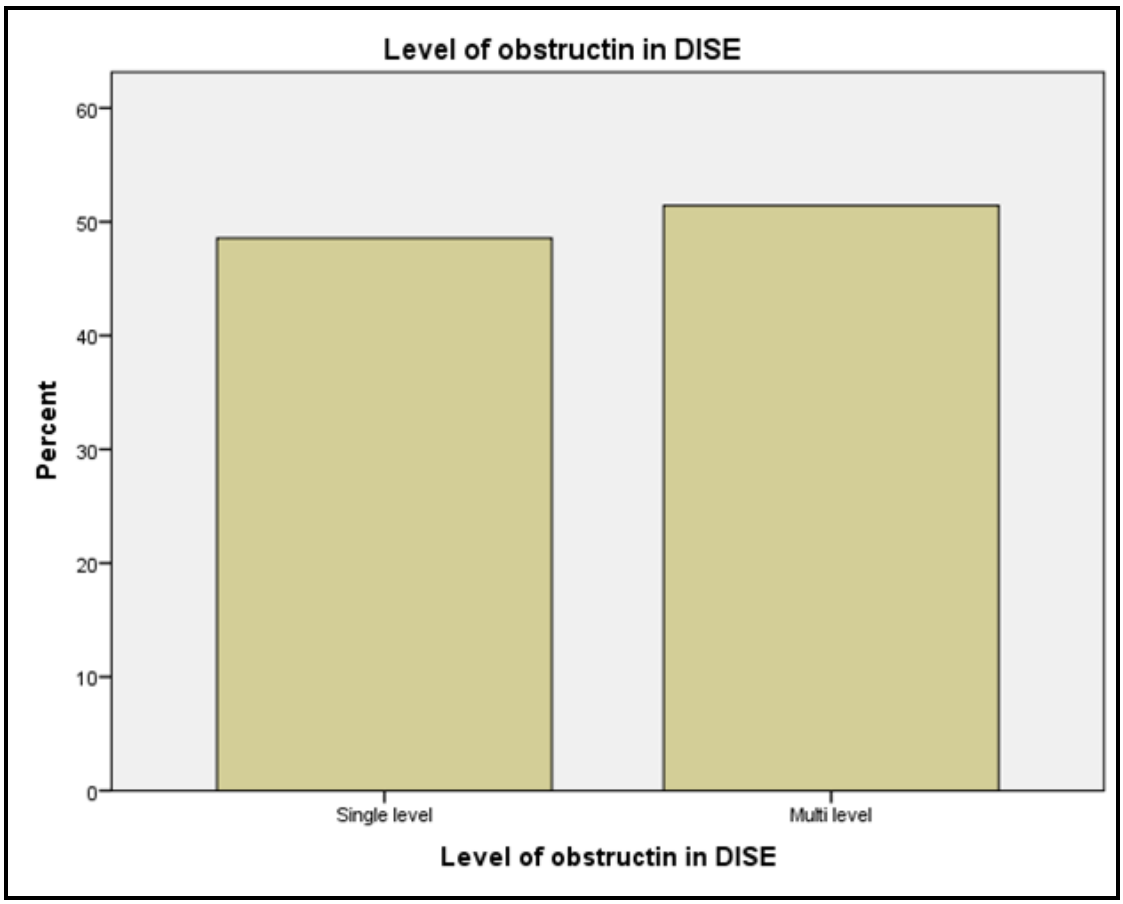
42.9% patients had single level obstruction.

48.6% patients had multilevel obstruction.



LEVEL OF OBSTRUCTION IN DISE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Single level	17	48.6	48.6	48.6
	Multi level	18	51.4	51.4	100.0
	Total	35	100.0	100.0	



48.6% patients had single level obstruction.

51.4% patients had multilevel obstruction.

AHI - AGE CROSSTABULATION

			Age			Total
			Upto 30 years	30 to 40 years	40 to 50 years	
AHI	Mild	Count	0	1	1	2
		% within AHI	.0%	50.0%	50.0%	100.0%
	Moderate	Count	3	6	2	11
		% within AHI	27.3%	54.5%	18.2%	100.0%
	Severe	Count	1	12	9	22
		% within AHI	4.5%	54.5%	41%	100.0%
Total		Count	4	19	12	35
		% within AHI	11.4%	54.3%	34.3%	100.0%

CHI-SQUARE TESTS

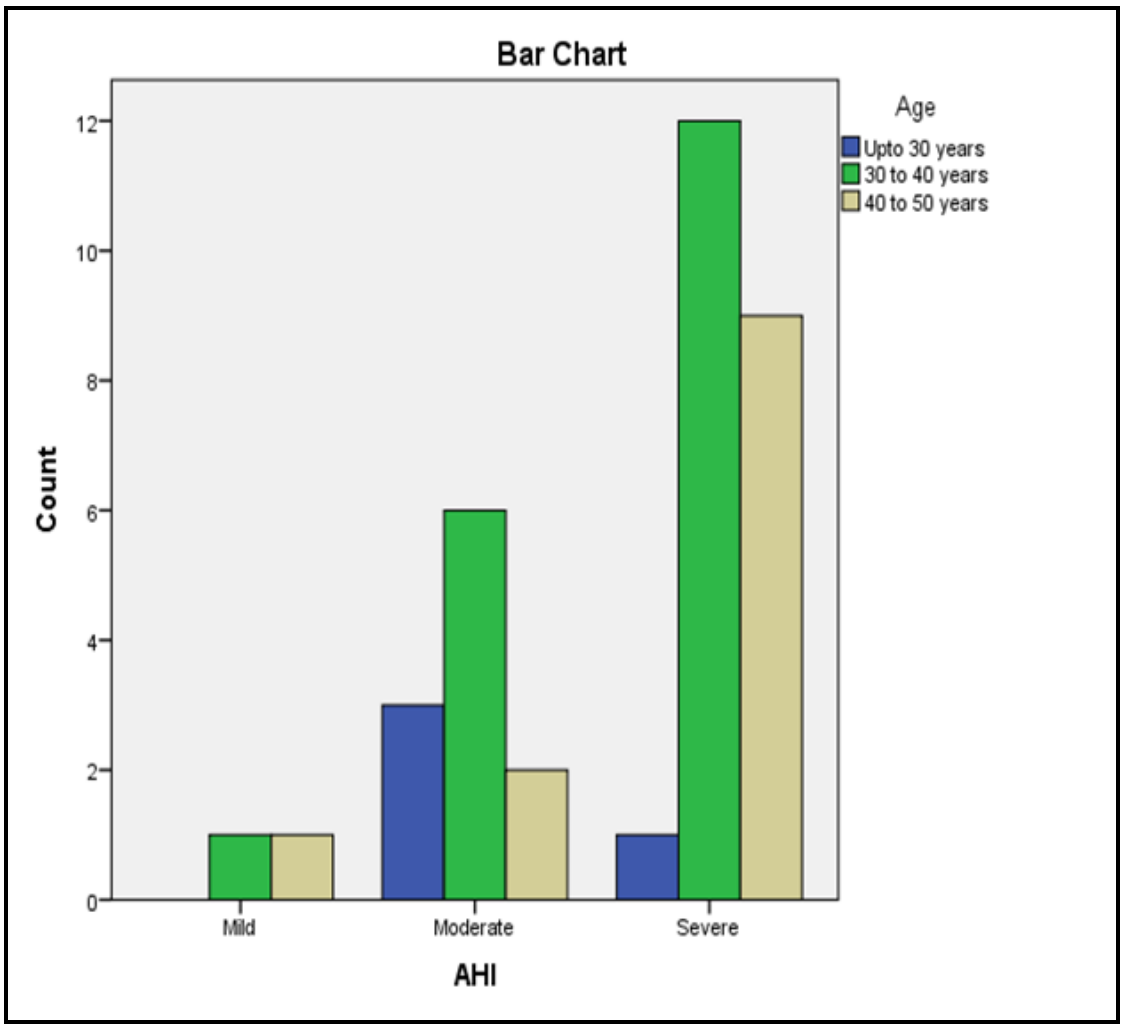
	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.822a	4	.306
Likelihood Ratio	4.779	4	.311
Linear-by-Linear Association	1.126	1	.289
N of Valid Cases	35		

a. 6 cells (66.7%) have expected count less than 5. The minimum expected count is .23

The above table shows thatof the 2 patients in mild OSA group 50% belong to 30-40 yrs and another 50% belongs to 40-50 yr age group.

Of 11 patients with moderate OSA 27.3%(3)belong to 20-30 yrs,54.5%(6)belong to 30-40 yrs and 18.2%(2) belong to 40-50 yrs.

Of 22 patients with severe OSA 4.5%(1)belong to 20=30 yrs,54.5%(12)belong to 30-40 yrs 41%belong to 40-50 yrs.



MEANSPO2 – AGE

CROSSTAB

			Age			Total
			Upto 30 years	30 to 40 years	40 to 50 years	
MEAN SPO2	Abnormal	Count	3	6	4	13
		% within MEANSPO2	23.1%	46.2%	30.8%	100.0%
	Normal	Count	1	13	8	22
		% within MEANSPO2	4.5%	59.1%	36.4%	100.0%
Total		Count	4	19	12	35
		% within MEANSPO2	11.4%	54.3%	34.3%	100.0%

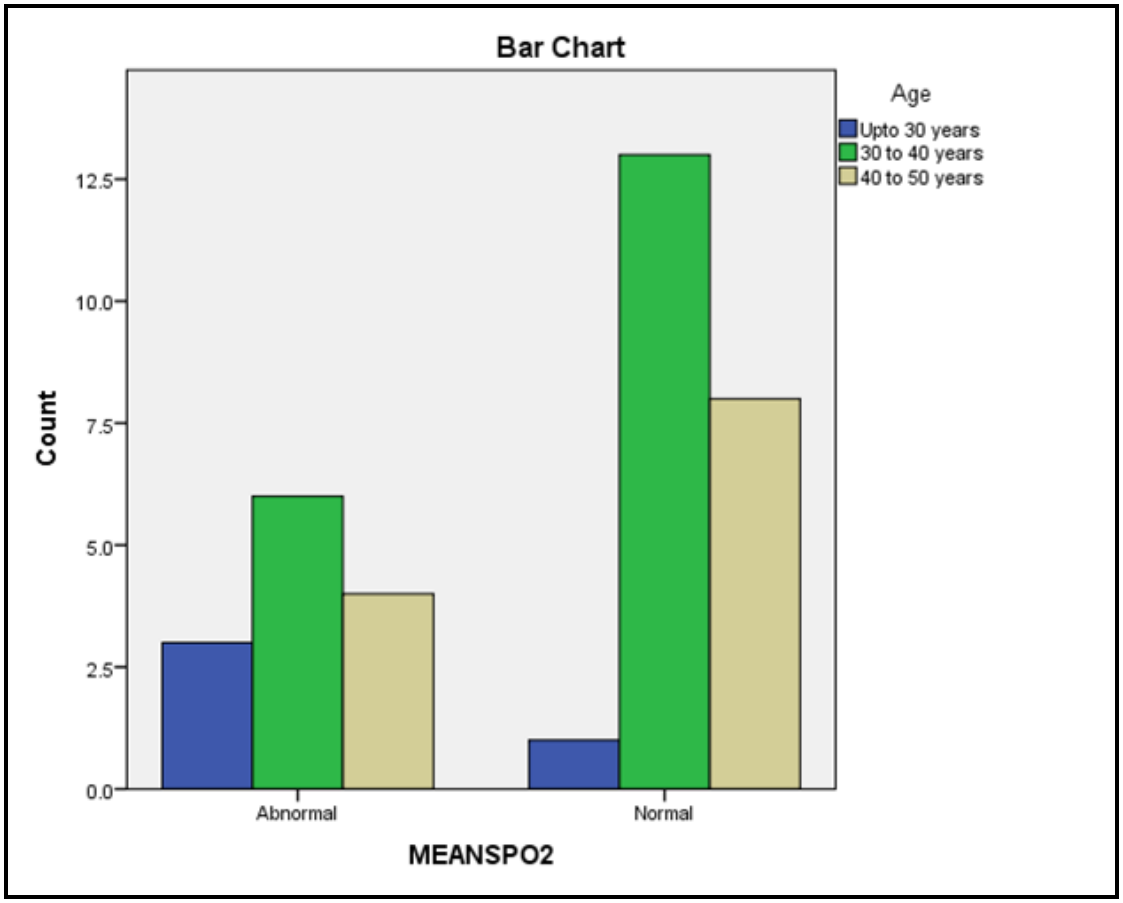
CHI-SQUARE TESTS

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.782a	2	.249
Likelihood Ratio	2.706	2	.258
Linear-by-Linear Association	1.141	1	.285
N of Valid Cases	35		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is 1.49.

Of 13 patients who had mean spo2 <90% 23.1%(3) belonged to <30 yrs,46.2%(6) belonged to 30-40 yrs 30.8%(4) belonged to 40-50 yrs.

Of 22 patients who had mean spo2>90% 4.5%(1) beloned to <30 yrs,59.1(13) belonged to 30-40 yrs,36.4%(8) belonged to 40-50 yrs


Bar Chart
Age
Upto 30 years
30 to 40 years
40 to 50 years
Count
12.5
10.0
7.5
5.0
2.5
0.0
Abnormal
Normal
MEANSPO2

LENGTH OF SOFT PALATE – AGE

CROSSTAB

			Age			Total
			Upto 30 years	30 to 40 years	40 to 50 years	
Length of soft palate	Normal	Count	1	9	2	12
		% within Length of soft palate	8.3%	75.0%	16.7%	100.0%
	Abnormal	Count	3	10	10	23
		% within Length of soft palate	13.0%	43.5%	43.5%	100.0%
Total		Count	4	19	12	35
		% within Length of soft palate	11.4%	54.3%	34.3%	100.0%

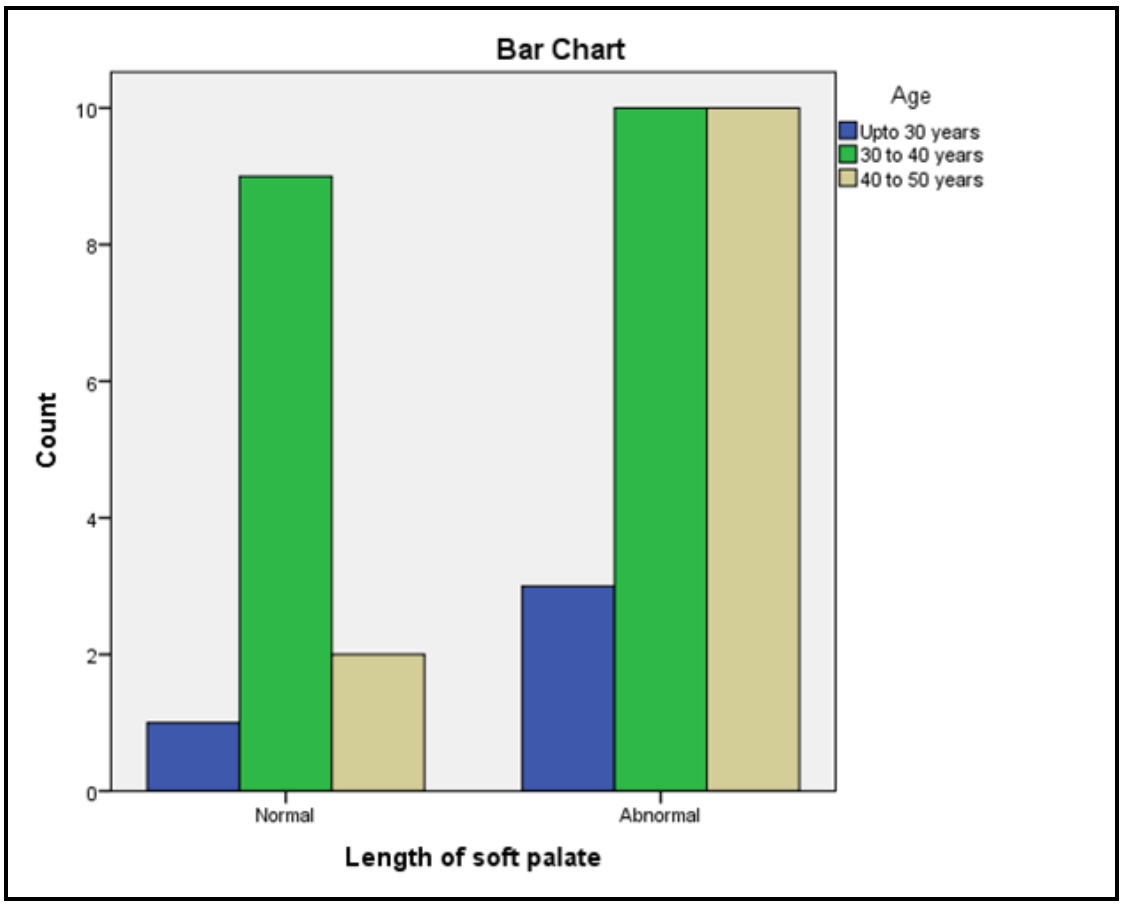
CHI-SQUARE TESTS

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	3.250a	2	.197
Likelihood Ratio	3.405	2	.182
Linear-by-Linear Association	.924	1	.336
N of Valid Cases	35		

3 cells (50.0%) have expected count less than 5. The minimum expected count is 1.37.

of 12 patients who had normal soft palate length(<3.5cm) 8.3%(1) belonged to <30 yrs,75%(9) belonged to 30-40 yrs,16.7%(2) belonged to 40-50 yrs.

Of 23 patients who had abnormal soft palate length 13%(3) belonged to <30 yrs,43.5%(10) belonged to 30-40 yrs,43.5%(10) belonged to 40-50 yrs.



THICKNESS OF SOFT PALATE - AGE

CROSSTAB

			Age			Total
			Upto 30 years	30 to 40 years	40 to 50 years	
Thickness of soft palate	Normal	Count	2	6	5	13
		% within Thickness of soft palate	15.4%	46.2%	38.5%	100.0%
	Abnormal	Count	2	13	7	22
		% within Thickness of soft palate	9.1%	59.1%	31.8%	100.0%
Total		Count	4	19	12	35
		% within Thickness of soft palate	11.4%	54.3%	34.3%	100.0%

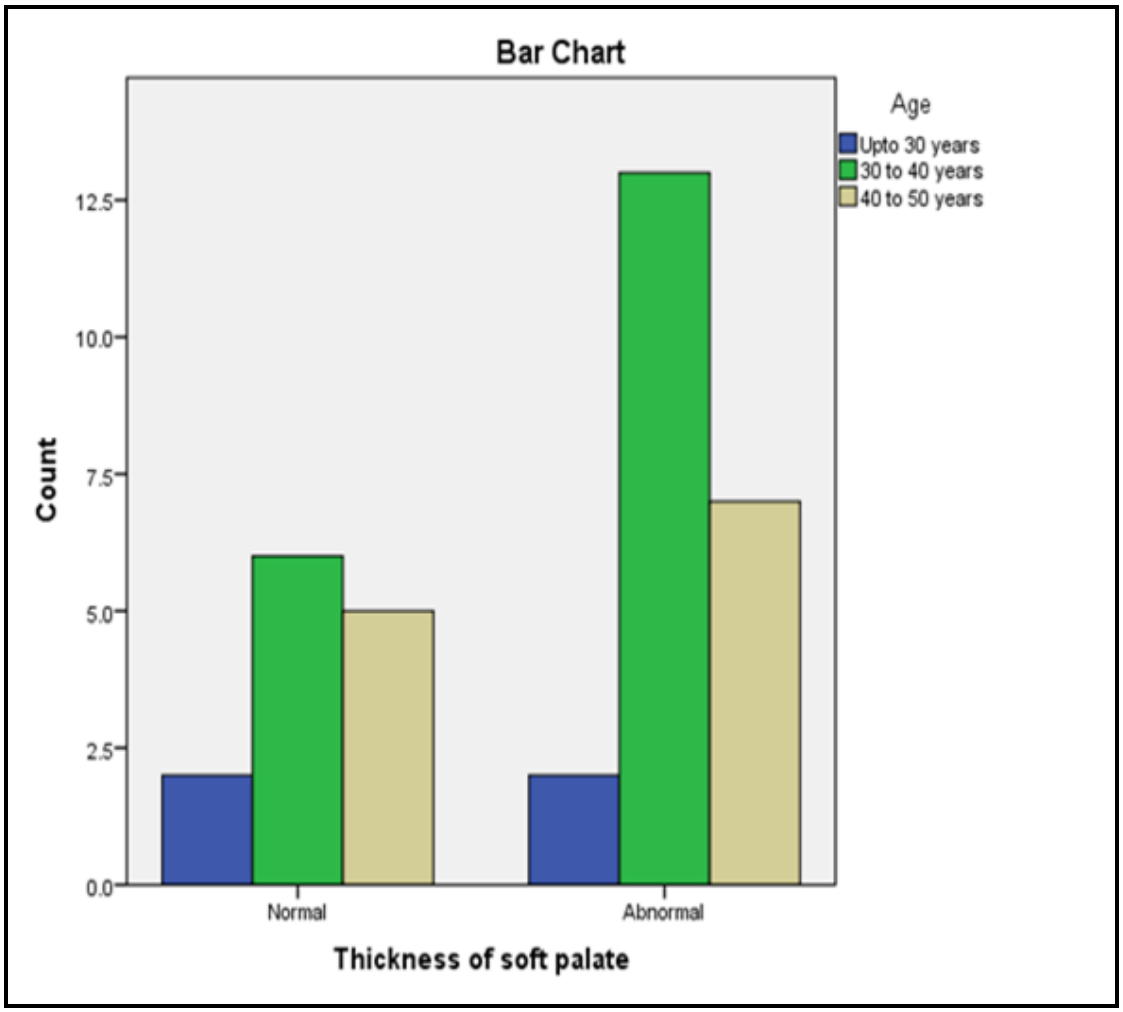
CHI-SQUARE TESTS

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.640a	2	.726
Likelihood Ratio	.635	2	.728
Linear-by-Linear Association	.000	1	.988
N of Valid Cases	35		

3 cells (50.0%) have expected count less than 5. The minimum expected count is 1.49.

0f 13 patients who had normal soft palate thickness (<1cm)15.4%(2) belonged to <30 yrs,46.2%(6) belonged to 30-40 yrs,38.5%(5) belonged to 40-50 yrs.

Of 22 patients who had abnormal soft palate thickness 9.15%(2) belonged to <30 yrs,59.1%(13) belonged to 30-40 yrs 31.8%(7) belonged to 40-50 yrs.



GRADING – AGE

CROSSTAB

			Age			Total
			Upto 30 years	30 to 40 years	40 to 50 years	
Grading	Palate obst	Count	1	7	4	12
		% within Age	25.0%	36.8%	33.3%	34.3%
	Palate and Oro	Count	2	6	1	9
		% within Age	50.0%	31.6%	8.3%	25.7%
	Multi segment	Count	0	2	0	2
		% within Age	.0%	10.5%	.0%	5.7%
	Tongue base	Count	0	2	2	4
		% within Age	.0%	10.5%	16.7%	11.4%
	Epiglottis	Count	1	2	5	8
		% within Age	25.0%	10.5%	41.7%	22.9%
Total		Count	4	19	12	35
		% within Age	100.0%	100.0%	100.0%	100.0%

CHI-SQUARE TESTS

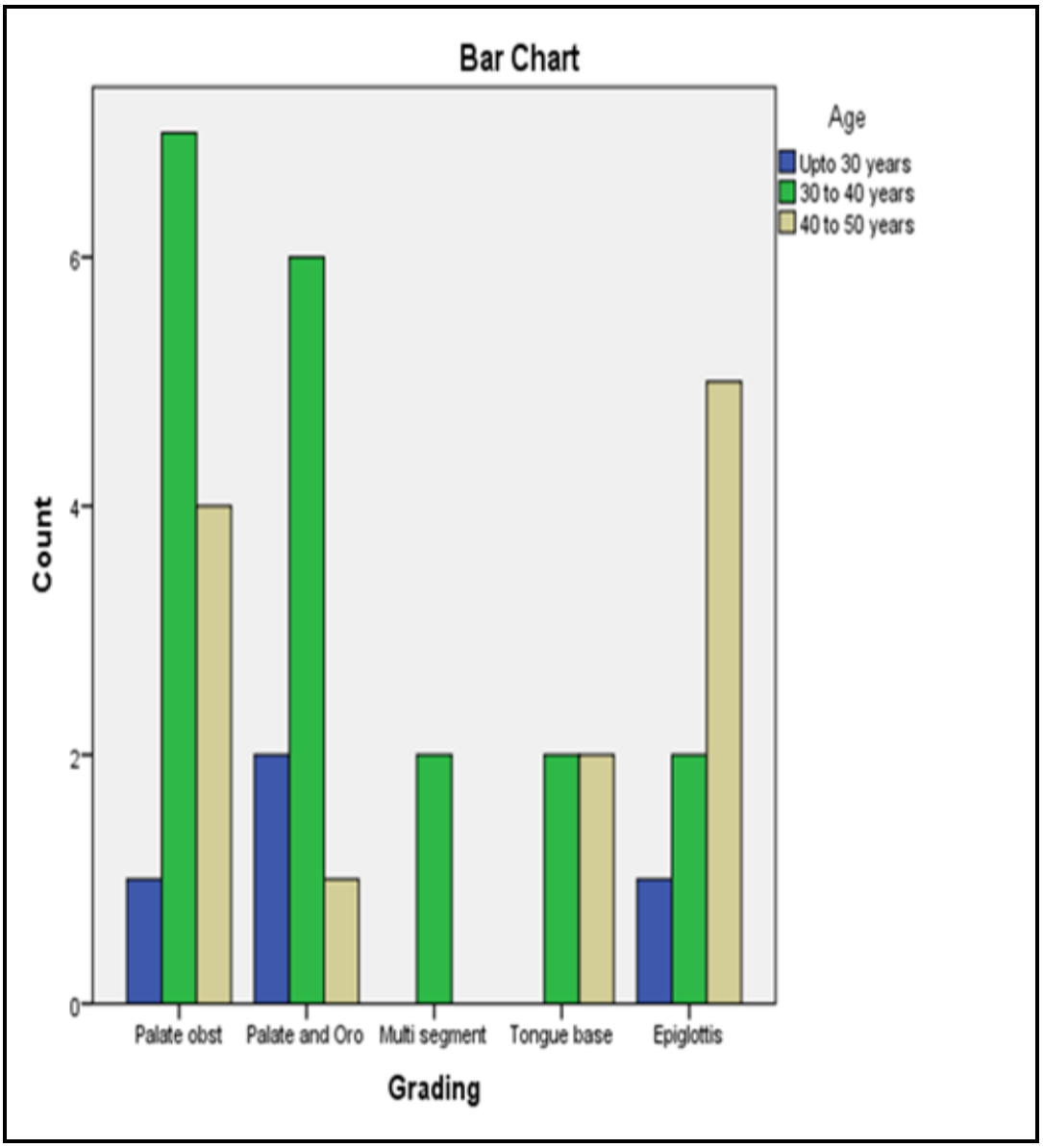
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	8.294a	8	.405
Likelihood Ratio	9.727	8	.285
Linear-by-Linear Association	1.729	1	.189
N of Valid Cases	35		

14 cells (93.3%) have expected count less than 5. The minimum expected count is .23.

Of 4 patients in upto 30 yrs age group 25%(1) had grade 2 obstruction in DISE,50%(2) had GRADE 3 obst,25%(1) had grade 6 obstruction.

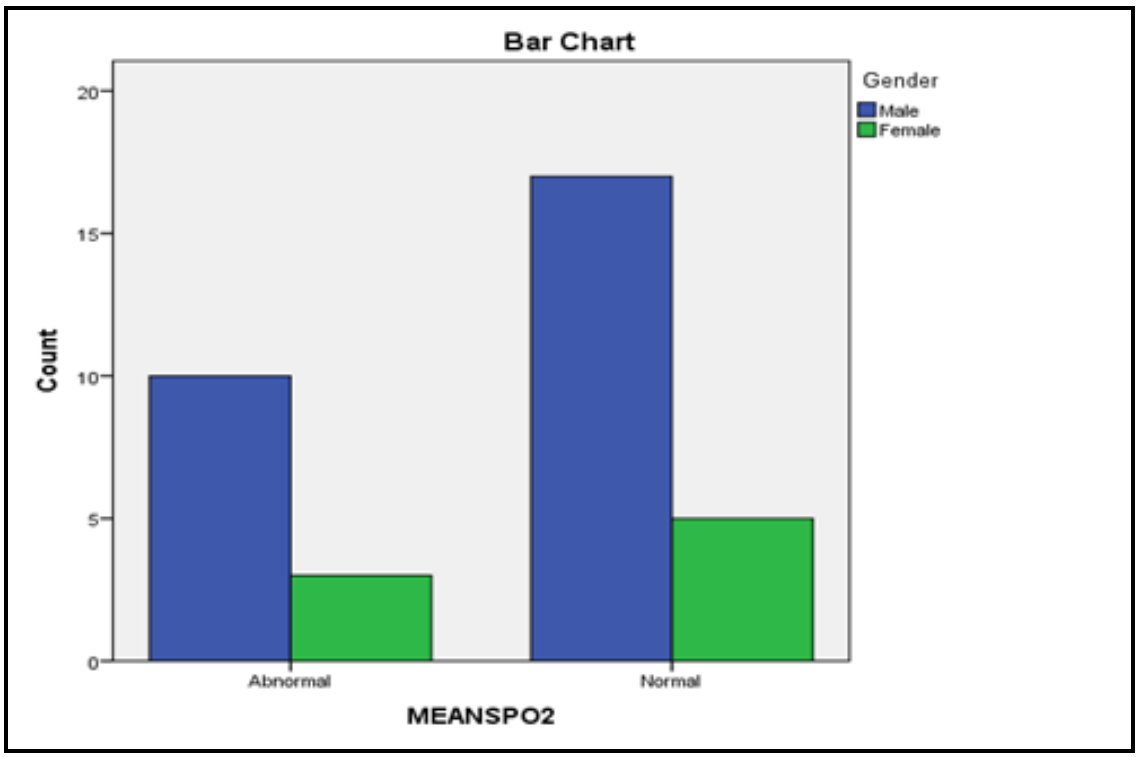
Of 19 patients in 30-40 yrs age group grade 2 obst-36.8%(7),grade 3-31.6%(6)grade 4-10.5%(2),grade 5-10.5%(2),grade 6-10.5%(2)

Of 12 patients in 40-50 yrs age group grade 2 obst-33.3%(4),grade 3-8.3%(1),grade 5-16.7%(2),grade 6-41.7%(5).



MEANSPO2 - GENDER CROSSTABULATION

			Gender		Total
			Male	Female	
MEAN SPO2	Abnormal	Count	10	3	13
		% within Gender	37.0%	37.5%	37.1%
	Normal	Count	17	5	22
		% within Gender	63.0%	62.5%	62.9%
Total		Count	27	8	35
		% within Gender	100.0%	100.0%	100.0%



Of 27 males 63%(17) had spo2>90%

Of 8 females 62.5%(5) had spo2>90%

AHI - GENDER CROSSTABULATION

			Gender		Total
			Male	Female	
AHI	Mild	Count	1	1	2
		% within Gender	3.7%	12.5%	5.7%
	Moderate	Count	6	5	11
		% within Gender	22.2%	62.5%	31.4%
	Severe	Count	20	2	22
		% within Gender	74.1%	25.0%	62.9%
Total		Count	27	8	35
		% within Gender	100.0%	100.0%	100.0%

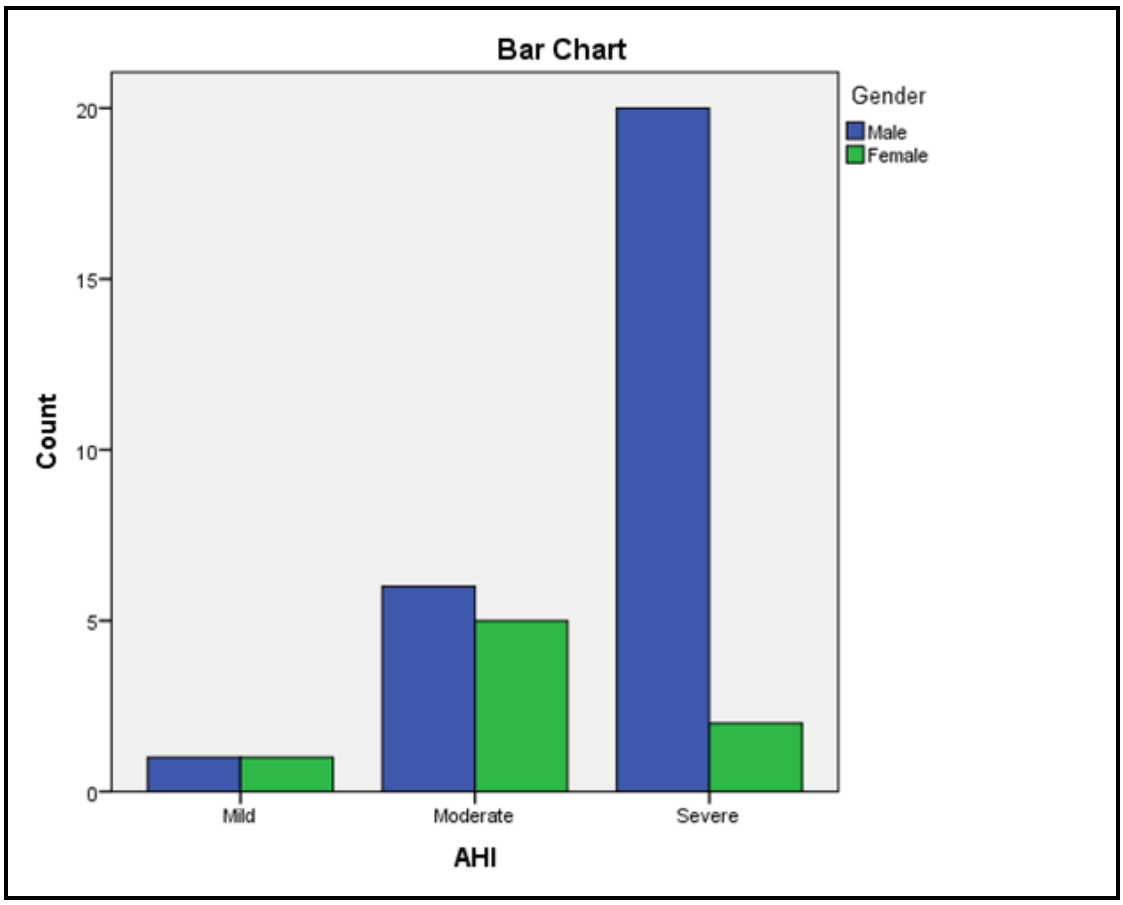
CHI-SQUARE TESTS

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	6.386a	2	.041
Likelihood Ratio	6.293	2	.043
Linear-by-Linear Association	5.590	1	.018
N of Valid Cases	35		

3 cells (50.0%) have expected count less than 5. The minimum expected count is .46.

From the above table there is a clear significance between AHI and gender.

Males have severe OSA (75%) compared to females.


Bar Chart
Gender
Male
Female
Count
20
15
10
5
0
Mild
Moderate
Severe
AHI

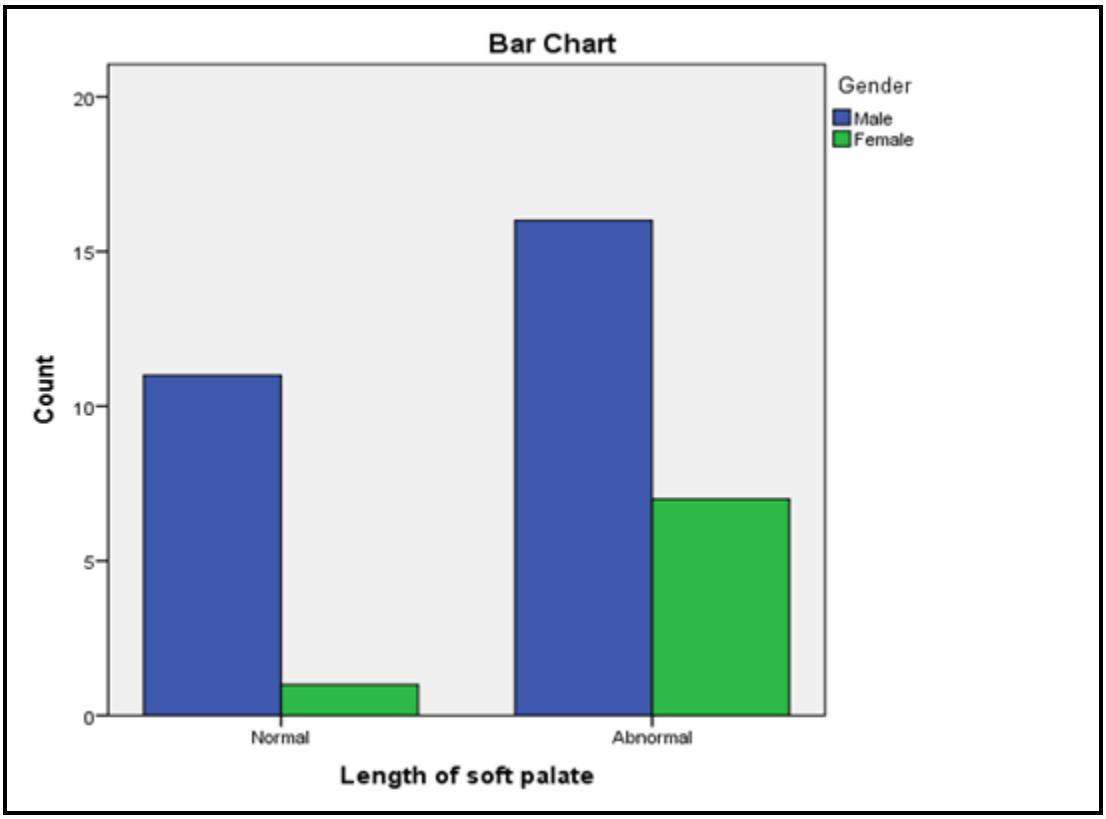
LENGTH OF SOFT PALATE – GENDER

CROSSTAB

			Gender		Total
			Male	Female	
Length of soft palate	Normal	Count	11	1	12
		% within Gender	40.7%	12.5%	34.3%
	Abnormal	Count	16	7	23
		% within Gender	59.3%	87.5%	65.7%
Total		Count	27	8	35
		% within Gender	100.0%	100.0%	100.0%

Of 27 males in the study 59.3%(16) had soft palate length>3.5cm

Of 8 females in the study 87.5% (7) had soft palate length >3.5cm



THICKNESS OF SOFT PALATE – GENDER

CROSSTAB

			Gender		Total
			Male	Female	
Thickness of soft palate	Normal	Count	10	3	13
		% within Gender	37.0%	37.5%	37.1%
	Abnormal	Count	17	5	22
		% within Gender	63.0%	62.5%	62.9%
Total		Count	27	8	35
		% within Gender	100.0%	100.0%	100.0%

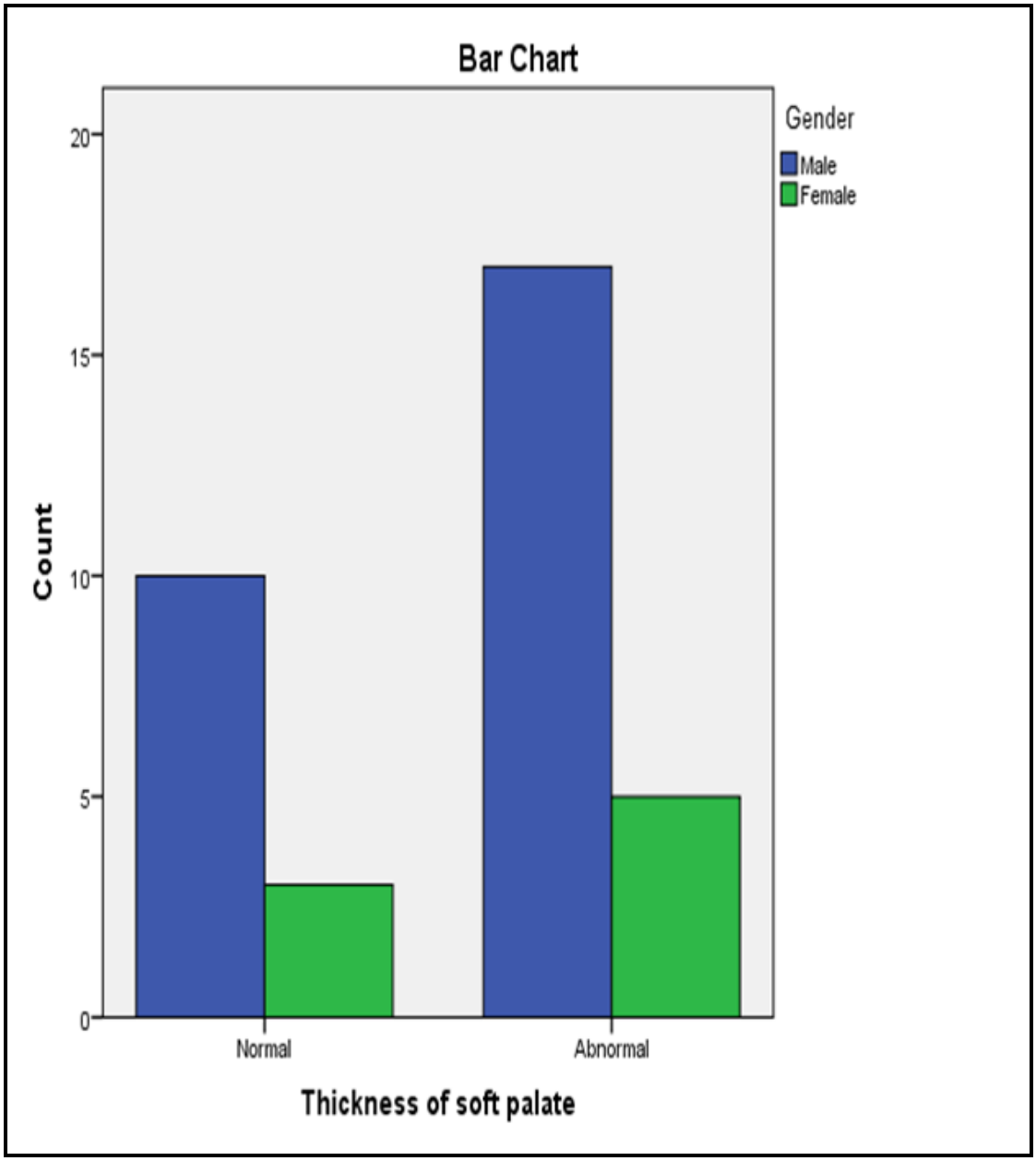
CHI-SQUARE TESTS

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.001a	1	.981		
Continuity Correctionb	.000	1	1.000		
Likelihood Ratio	.001	1	.981		
Fisher's Exact Test				1.000	.645
Linear-by-Linear Association	.001	1	.981		
N of Valid Cases	35				

a. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 2.97.

b. Computed only for a 2x2 table

63% of males had soft palate thickness>1cm.


Bar Chart
Gender
Male
Female
Count
20
15
10
5
0
Normal
Abnormal
Thickness of soft palate

LEVEL OF OBSTRUCTION IN MRI – GENDER

CROSSTAB

			Gender		Total
			Male	Female	
Level of obstruction in MRI	No obstruction	Count	3	0	3
		% within Gender	11.1%	.0%	8.6%
	Single level	Count	11	4	15
		% within Gender	40.7%	50.0%	42.9%
	Multi level	Count	13	4	17
		% within Gender	48.1%	50.0%	48.6%
Total		Count	27	8	35
		% within Gender	100.0%	100.0%	100.0%

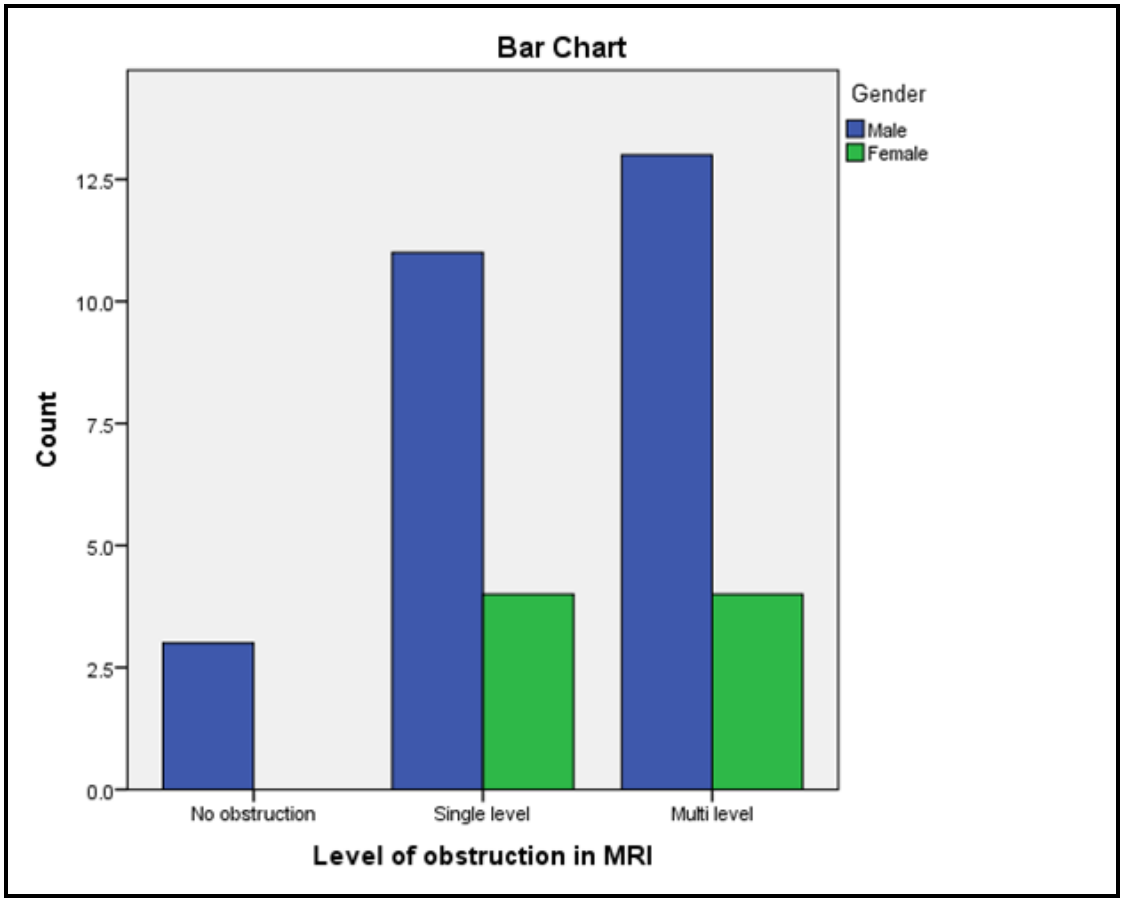
CHI-SQUARE TESTS

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.017a	2	.601
Likelihood Ratio	1.680	2	.432
Linear-by-Linear Association	.245	1	.621
N of Valid Cases	35		

a. 4 cells (66.7%) have expected count less than 5. The minimum expected count is .69.

48.1% males (13) had multilevel obstruction in dynamic MRI.

50% females (4) had multilevel obstruction in MRI.


Bar Chart
Gender
Male
Female
Count
12.5
10.0
7.5
5.0
2.5
0.0
No obstruction
Single level
Multi level
Level of obstruction in MRI

LEVEL OF OBSTRUCTIN IN DISE – GENDER

CROSSTAB

			Gender		Total
			Male	Female	
Level of obstructin in DISE	Single level	Count	13	4	17
		% within Gender	48.1%	50.0%	48.6%
	Multi level	Count	14	4	18
		% within Gender	51.9%	50.0%	51.4%
Total		Count	27	8	35
		% within Gender	100.0%	100.0%	100.0%

CHI-SQUARE TESTS

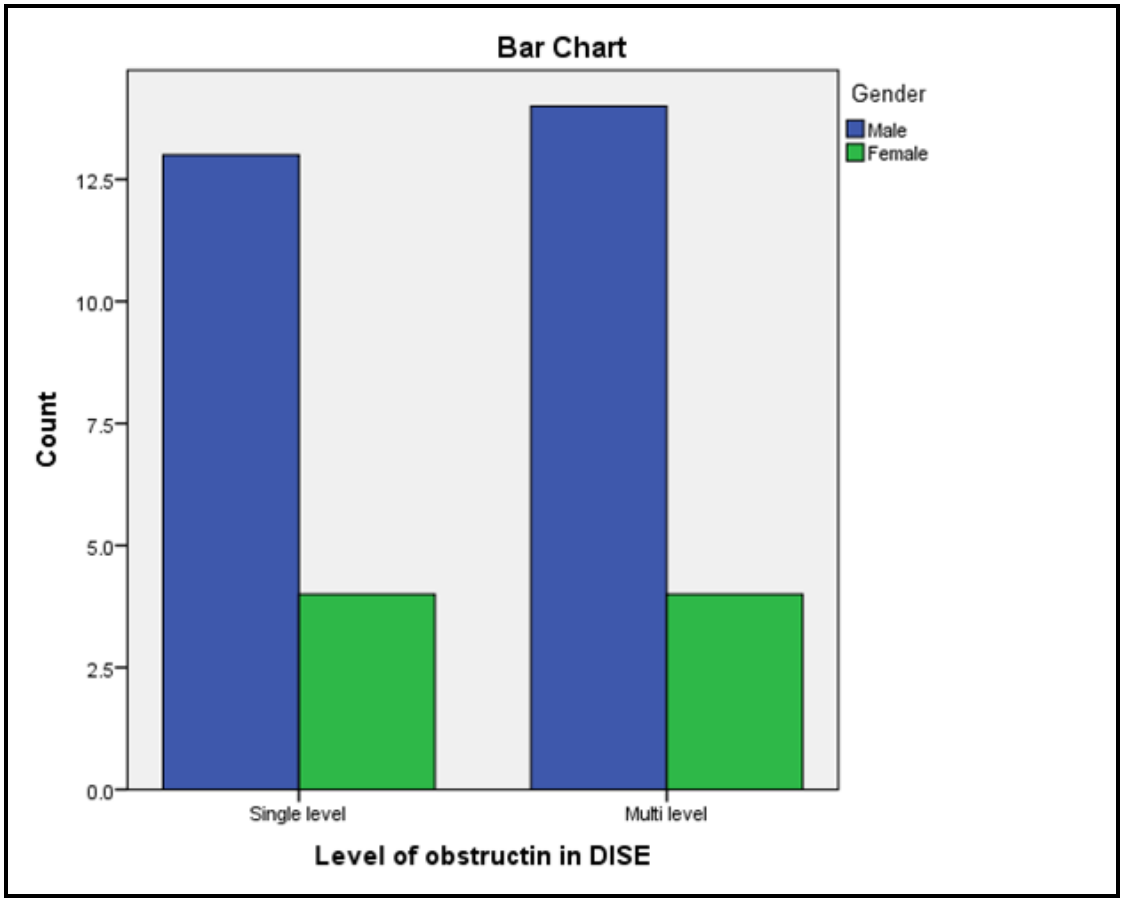
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.008a	1	.927		
Continuity Correctionb	.000	1	1.000		
Likelihood Ratio	.008	1	.927		
Fisher's Exact Test				1.000	.620
Linear-by-Linear Association	.008	1	.928		
N of Valid Cases	35				

a. 2 cells (50.0%) have expected count less than 5. The minimum expected count is 3.89.

b. Computed only for a 2x2 table

51.9% males had multilevel obstruction in DISE.

50% females had multilevel obstruction in DISE.


Bar Chart
Gender
Male
Female
Count
12.5
10.0
7.5
5.0
2.5
0.0
Single level
Multi level
Level of obstructin in DISE

DISCUSSION

The study was started with the aim of comparing the levels of obstruction in dynamic MRI and DISE in OSA and snoring patients. The study population chosen was scrutinized with proper implementation of the inclusion criteria. The confounding factors like other systemic disorders are excluded.

REVIEW OF LITERATURE

1.Marais compared " the presence or absence of snoring and its site of generation between a group of 205snorers and another of 126 non-snorers. Snoring was produced at nasendoscopy in 45.3% of non-snorers but could not be produced in 18.1% of snorers. There was no significant difference in the site of sound production between the two groups and although the noise produced by the non snoring group was quieter, this difference was not significant".(19)

2.Abdullah et al. "suggested that a clear establishmentof the site of obstruction is crucial for subsequent treatment planning". Video sleep nasendoscopy (VSE) is probablythe most accurate assessment of the situation and also helps in identifying the situation that needs correction.(31)

3.Berry et al. conducted " a prospective cohort study involving 107 patients divided into two groups. The twogroups of patients were matched for their Body Mass Index (BMI). The first group consisted of 53 patients with a history suggestive of obstructive sleep apnea. The second group consisted of 54 patients with a partner-confirmed history of no snoring. These patients

were undergoing anesthesia for other reasons. Both groups of patients were free of associated otorhinolaryngological symptoms. The main outcome measure was assessment of production of snoring or obstruction in patients with no documented history of snoring when sedation was administered as part of genera lanesthesia using TCI with propofol. Both groups contained similar numbers of males and females but there was a predominance of males in the snoring group whereas females were predominant in the non snoring group.All patients in the symptomatic (snoring) group snored or obstructed at different concentrations of propofol. There was no statistically significant difference($P = 0.401$) in the distributions of concentrations of propofol at which snoring started between men and women.In comparison, all patients in the asymptomatic (nonsnoring)group could not be induced to snore or obstruct at incremental levels of propofol and this was clearly significant statistically ($P < 0.001$)".(15)

4.**oliver M. vandervekan** et al "conducted a study to identify the possible predictive value of drug-induced sleep endoscopy (DISE) in assessing therapeutic response to implanted upper airway stimulation (UAS) for obstructive sleep apnea (OSA) and concluded that the absence of palatal Complete concentric collapse during DISE may predict therapeutic success with implanted UAS therapy."(32)

5.Yuji suto et al. observed the usefulness of cine mri in OSAS.Fifteen patients with sleep apnea and five healthy volunteers underwent ultrafast MR imaging while awake and during sleep . Sequential midline sagittal images of the pharynx were obtained and displayed in the cine mode Patients with sleep

apnea were found to have sites of pharyngeal abnormality that were not present in healthy volunteers. Nine sites of narrowing in seven patients (47%) were detected with the patient awake; 21 sites of obstruction in 13patients (87%) were diagnosed with the patient asleep. Six patients showed only one obstruction, and seven had several obstructions.(29)

6.Anacelia Faria et al "conducted a prospective study of thirty-two patients with a polysomnographic diagnosis of OSA. All patients were submitted to MR imaging in order to obtain high-definition anatomical sagittal sequences during wakefulness and during sleep induced with Propofol. An area was defined on the sagittal plane in the midline of the pharynx. This region was called pharyngeal mid plane (PMP) area. A significant difference in PMP area (mm2) was observed between wakefulness and induced sleep in each patient ($p < 0.000001$).

The patients with OSA suffer a significant reduction of 75,5 % in the area of the pharynx during induced sleep compared to wakefulness."(30)

RESULTS

Out of 35 patients in the study 29 patients had same levels of obstruction in both dynamic MRI and DISE. There is 82.8% correlation between dynamic MRI and DISE in identifying levels of obstruction.

Of the 6 patients who had different results in MRI and DISE **3 patients** had no obstruction in Dynamic MRI but found to have obstruction at VELUM in DISE.

ONE patient who had tongue base obstruction in Dynamic MRI had tongue base and epiglottis level obstruction in DISE.

ONE patient had obstruction in velum and epiglottis in Dynamic MRI but had obstruction only in velum level in DISE.

ONE patient had obstruction at oropharynx in Dynamic MRI was found to have obstruction in oropharynx and epiglottis in DISE.

CONCLUSION

OSAS is a disease of modern ages and identified as distinct entity for past 20 to 25 years .At present ,OSAS has been identified as a separate risk factor or an entity for increased susceptibility to stroke, myocardial infarction, cardiac arrhythmias, hypertension, dyslipidemia, insulin resistance and diabetes mellitus, depression and sexual dysfunction. It is very important to identify the level of obstruction to decide the treatment protocol. Level of obstruction can be at velum, oropharynx, tongue base,epiglottis.This can be identified with Dynamic MRI and DISE. Dynamic MRI is non invasive procedure with no radiation hazards and gives accurate measurements of upper airway at velum,oropharynx and hypopharynx levels and it is done under normal sleep ,whereas in DISE patients are subjected to anaesthesia induced sleep and there can be subjective variations in quantifying the results .CLAUSTROPHOBIA is the main disadvantage in MRI. DISE gives equally good results as dynamic MRI, the only problem being subjecting the patient to an anasthetic procedure. Since Dynamic MRI is non invasive and gives accurate measurements of upper airway ,Dynamic MRI is superior to DISE in identifying the level of obstruction. In all cases planned for surgery after identifying level of obstruction with dynamic MRI,DISE can be done on table before the planned surgery to confirm the level of obstruction so that separate anesthesia for DISE can be avoided.

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10) Friedman's sleep apnea and snoring: surgical and non-surgical therapy chapter 12 table 12.1 pg 79

11) Friedman’s sleep apnea and snoring: surgical and non-surgical therapy chapter 12 table 12.3 pg 81

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ANNEXURES

Epworth sleepiness scale

Patients are asked to grade from 0 to 3 their likelihood of falling asleep in contrast to just feeling tired in certain situations.

The total score will be between **0 and 24.**

Level of sleepiness

0 = would never doze

1 = slight chance of dozing

2 = moderate chance of dozing

3 = high chance of dozing

Situations:

1. Sitting and reading
2. Watching TV
3. Sitting inactive in a public place (theater, meeting)
4. As a passenger in a car for 1 h without a break
5. Lying down to rest in the afternoon
6. Sitting and talking someone
7. Sitting quickly after lunch (without alcohol)
8. In a car while stopped in traffic

PROFORMA

Name : **Date :**

Age : **Ip / Op no :**

Sex :

Occupation :

Presenting complaints :

History of presenting complaints :

S.no.	Complaints	yes	no	duration
1.	History of snoring			
2.	History of sleep awakening / insomnia			
3.	History of excessive day time sleepiness			
4.	History of impaired cognitive function			
5.	History of Hallucinations			
6.	History of increased weight gain			
7.	History of choking in sleep			
8.	History of nasal obstruction			
9.	History of mouth breathing			

Past history : Diabetes mellitus / Hypertension / Epilepsy / Asthma / Jaundice / previous history of accidents

Personal history : Diet , appetite , smoking , alcohol , chronic drug intake , bowel and bladder habits

Family history : married / unmarried

General examination :

Built :

Height :

Weight :

BMI :

Neck circumference :

Waist circumference :

Temperature ;

Pallor :

Cyanosis :

Jaundice :

Pedal edema :

Vitals ;

Pulse : BP : Respiratory rate :

Systemic examination :

Respiratory system :

Cardiovascular system :

Central nervous system :

ENT Examination :

Throat :

Oral cavity :

Gums :

Oral mucosa :

Floor of mouth :

Anterior 2/3rd tongue :

Hard and soft palate :

MALLAMPATI score :

Oropharynx :

Antr. Pillar :

Postr. Pillar :

Tonsil :

Posterior pharyngeal wall :

Indirect laryngoscopy :

Posterior 1/3rd tongue :

Vallecula :

Epiglottis :

Vocal cords and mobility :

Posterior pharyngeal wall :

Nose :

External contour :

Antr. Rhinoscopy :

Postr. Rhinoscopy :

Ear :

Pinna :

External auditory canal :

Tympanic membrane :

Neck :

Laryngeal contour :

Abnormal veins / scars :

Accessory muscles of respiration :

Investigations :

Blood investigations :

Thyroid functions test :

Diagnostic nasal endoscopy :

Video direct laryngoscopy :

x-ray Skull : Anteroposterior

Lateral

x-ray chest :

Patient name :

Diagnosis :

Management :

- Investigations
- Anesthesia
- Procedure

PATIENT CONSENT FORM

Title of the Project : A COMPARITIVE STUDY OF DYNAMIC MRI WITH DRUG INDUCED SLEEP ENDOSCOPY IN OBSTRUCTIVE SLEEP APNEA PATIENTS

Institution : Upgraded Institute of Otorhinolaryngology,
Madras Medical College,
Chennai – 600003.

Name : Date :

Age : IP No. :

Sex : Project Patient No. :

The details of the study have been provided to me in writing and explained to me in my own language.

I confirm that I have understood the above study and had the opportunity to ask questions.

I understood that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected.

I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

I have been given an information sheet giving details of the study.

I fully consent to participate in the above study.

_____	_____	_____
Name of the subject	Signature	Date
_____	_____	_____
Name of the Investigator	Signature	Date

IJL 1284.4
IJL

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.Chandru.R.
PG in MS (ENT)
Madras Medical College
Chennai 600 003

Dear Dr.Chandru.R.

The Institutional Ethics Committee has considered your request and approved your study titled **" A COMPARATIVE STUDY OF DYNAMIC M.R.I. WITH DRUG INDUCED SLEEP ENDOSCOPY IN OBSTRUCTIVE SLEEP APNEA" NO.31022015**

The following members of Ethics Committee were present in the meeting held on 03.02.2015 conducted at Madras Medical College, Chennai 3.

1. Dr.C.Rajendran, MD	:Chairperson
2. Dr.R.Vimala,MD.,Dean,MMC,Ch-3	: Deputy Chairperson
3. Dr.B.Kalaiselvi,MD.,Vice Principal,MMC,Ch-3	: Member Secretary
4. Dr..R.Nandhini,MD.,Inst.of Pharmacology,MMC	: Member
5. Dr..P.Ragumani, MS., Professor, Inst.of Surgery,MMC	: Member
6. Dr..K.Ramadevi, Director , Inst.of Bio-Chem.MMC	: Member
7. Dr..Saraswathy,MD.,Director,Pathology, MMC	: Member
8. Dr.Md.Ali, MD., DM.,Prof.&HOD of Medl.GE,MD.MMC	: Member
9: Dr.S.G.Sivachidambaram,Director I/c, Inst.of Internal Medicine	: Member
10. Thiru S.Rameshkumar	: Lay Person
11.Thiru S.Govindasamy, BA., BL.,	: Lawyer
12.Tmt.Arnold Saulina, MA., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary - Ethics Committee

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

Sys 2

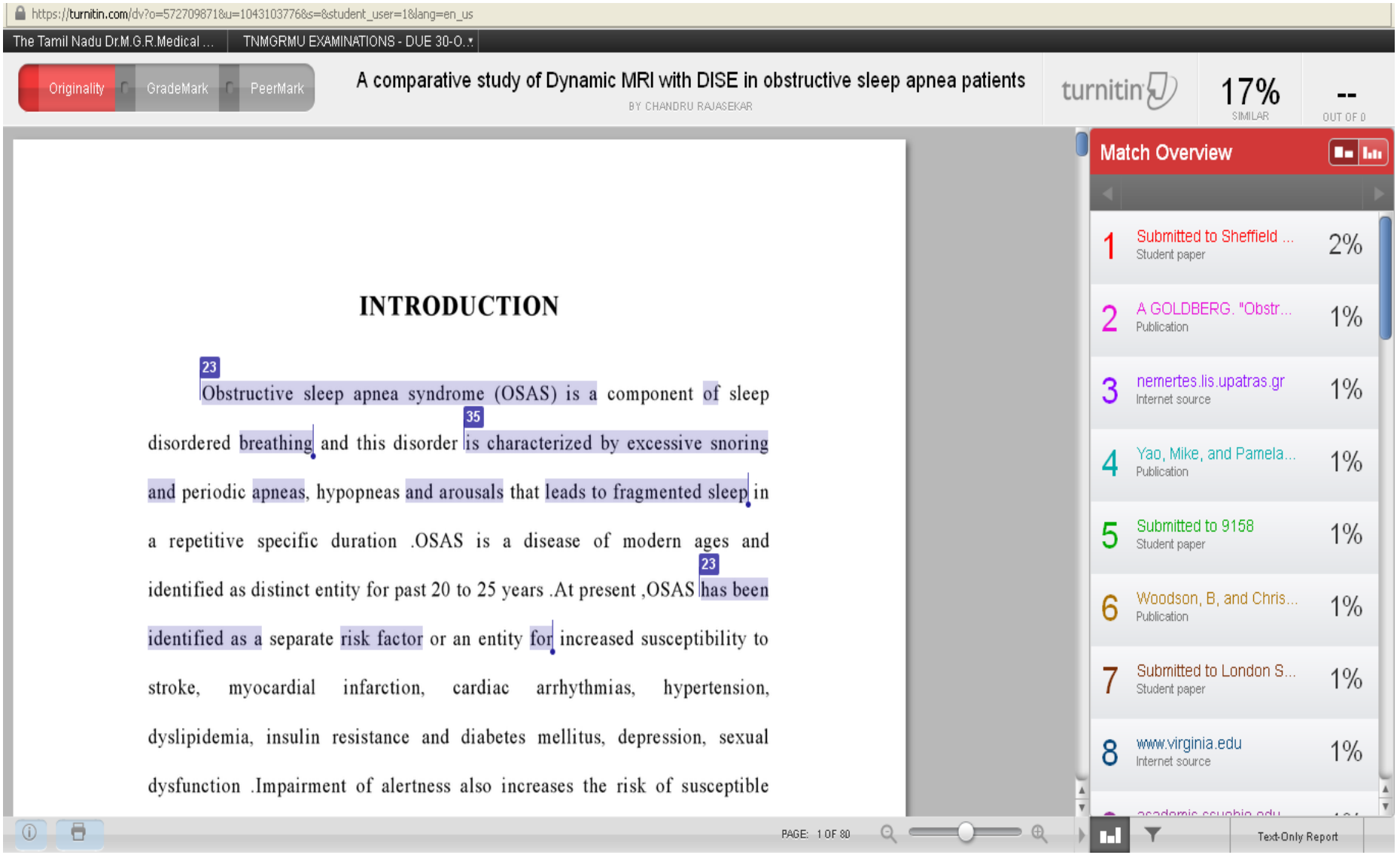
S.NO	NAME	AGE	SEX	POLYSOMNOGRAPHY							DYNAMIC MRI								DISE	
				CENTRAL APNOEA	OBSTRUCTIVE APNOEA	HYPNOEA	AHI	MEAN HR	MEAN SPO2	MIN SPO2	SOFT PALATE DIMENSIONS		AIRWAY DIMENSIONS						LEVEL OF OBSTRUTION	LEVEL OF OBSTRUCTION	GRADING
											LENGTH	THICK	AWAKE		MULLER		SLEEP				
													AP	LAT	AP	LAT	AP	LAT			
1.	MAHALAKSHMI	35	F	64	93	134	44.6	62.3	87	76	4.5	1.2	13	8.9	6.8	0.7	12.1	3.5	velum, oro	Velum ,oro	4
													14.7	14.3	5.6	4.3	6.7	5.4			
													14.5	12.4	11.4	13.5	11.3	12.5			
2.	MASTHAN BASHA	34	M	8	29	76	34.1	85.5	97	91	4	1.01	10.9	7.8	3.8	2.6	4.5	3.5	velum	velum	2
													8.9	14.3	4.5	7.8	5.4	8.6			
													13.5	12.7	6.8	4.5	7.5	5.4			
3.	KUMARAN	38	M	22	76	184	41.1	75.5	94	82	3.5	1.09	17.4	14	13.3	6.5	14.5	7.6	Velum, oro	Velum, oro	3
													14.5	12.3	7.6	4.5	8.7	6.6			
													13.5	13.4	8.6	6.5	9.6	5.8			
4.	PRABHU	36	M	43	90	48	51.5	77.6	93	73	2.9	0.9	10.1	16.3	12.4	15.5	13.4	16.7	Single oro	Multiple Oro, epiglottis	6
													8.3	13.4	4.5	6.7	6.7	4.5			
													13	24	17.8	23.4	7.8	11.5			
5.	PALANIVEL	37	M	9	7	146	28.5	81	92	78	3.4	1.2	4.5	7.8	0	0	1	0.5	Single velum	velum	2
													5.8	8.9	3.4	7.5	3.5	7.8			
													28	29	18.8	14.6	17.4	12.3			
6.	SHANKAR	38	M	13	171	161	47.7	68.9	93	81	3.5	0.9	8.3	10.6	3.8	2.8	4.2	3.5	Velum, oro	Velum, oro	3
													12.5	4.6	0	0	0	0			
													19.6	14.8	17.4	10.2	14.8	11.2			
7.	SRINIVAS	30	M	37	260	223	59.1	81.3	86	58	2.6	0.8	4.5	9.8	0.3	0.2	1.2	0.6	velum	velum	2
													4.2	9.7	7.6	5.6	4.5	4.6			
													24	22	18.3	14.4	17.8	12.7			
8	JINCE	32	M	12	102	121	32	81.3	92	86	2.4	1.1	8.1	10	4.5	3.8	4.7	3.9	Multiple Velum oro	Velum, oro	4
													11	3.5	0	0	0	0			
													19	14	17	10	14	11.2			
9.	KUMARA KRISHNAN	41	M	55	110	454	81.8	60	77	51	3.8	1	3.9	8.5	0	0	1.2	0.5	Single velum	velum	2
													3.9	8.2	3.4	7.5	3.5	7.8			
													24	22	18.8	14.2	17.8	12.2			
10.	PRASATH	35	M	12	102	128	32.6	69.4	92	86	4.2	1.1	3.7	8.9	0	0	1	0.9	Single velum	velum	2
													3.9	8.2	3.4	7.5	3.5	7.8			
													24	22	18	14	17	12			
11.	MADHESWARAN	48	M	74	64	254	59.8	87.9	91	79	3	0.9	13.7	11.7	5	9.7	6.4	8.6	Multiple Velum oro EPI	Velum, Oro EPI	6
													8.3	13.2	6.8	6.8	7.6	8.8			
													14.6	9.8	1.3	1.5	1.9	2.4			
12.	SOUNDARI	27	F	4	30	125	23.7	96.3	84	83	2.9	1	9.7	10.1	9.6	7.5	9.5	8.5	Single ORO	ORO	3
													7.9	13.4	5.6	7.7	4.7	6.6			
													14	26	9.8	22.4	8.8	21.3			
13.	VIJAY	28	M	34	28	214	25.4	67.5	93	81	4	1.2	10.1	16.3	12.4	15.5	13.4	16.7	No Obstruction	Velum	2
													10	13.9	12.6	8.9	11.5	9.7			
													13	24	17.8	23.4	16.9	21.9			
14.	RAMAMURTHY	37	M	22	88	125	33.1	96.5	90	68	4.5	1.1	8.1	10	4.5	3.8	4.7	3.9	Multiple Velum oro	Velum, Oro	3
													11	3.5	0	0	0	0			
													19	14	17	10	14	11.2			
15.	ELLAPPAN	39	M	5	114	271	42	100.2	81	60	2.5	0.4	13.2	17.4	12.3	15.4	13.1	16.3	No Obstruction	Velum	2
													14.4	10.5	12.6	8.8	11.2	9.5			

S.NO	NAME	AGE	SEX	POLYSOMNOGRAPHY							DYNAMIC MRI								DISE	
				CENTRAL APNOEA	OBSTRUCTIVE APNOEA	HYPNOEA	AHI	MEAN HR	MEAN SPO2	MIN SPO2	SOFT PALATE DIMENSIONS		AIRWAY DIMENSIONS						LEVEL OF OBSTRUTION	LEVEL OF OBSTRUCTION	GRADING
											LENGTH	THICK	AWAKE		MULLER		SLEEP				
													AP	LAT	AP	LAT	AP	LAT			
													18.4	25.6	17.7	23.8	16.3	21.2			
16.	DEEPAK KUMAR	42	M	27	85	116	32.4	74.5	91	86	4.4	0.9	3.7	8.9	0	0	1	0.9	Single velum	velum	2
													3.9	8.2	3.4	7.5	3.5	7.8			
													24	22	18	4	17	12			
17.	KUMAR	49	M	43	29	259	44.9	73.8	93	79	3.8	1	21	22	20.3	21.6	21.1	23.4	Tongue base	Tongue base	5
													17.4	19.8	9.4	7.8	10.2	6.4			
	SURESH KUMAR	39	M	90	85	232	48.8	84.7	86	67	2.7	0.9	13.2	13.5	5	5.5	6.5	7.2	Multiple Velum EPI	SINGLE VELUM	2
													12.1	12.1	4.9	8.5	5.2	7.8			
													14.7	15.8	1.2	1.3	2.3	3.5			
19.	THANGARAJ	44	M	7	52	81	15.1	78.8	95	83	5	1.3	16	24	14	16	15	15.4	Single Tongue Base	Tongue Base and EPI	6
													9.1	13.6	0	0	1.3	1.2			
													21	38	7	35	9.8	24			
20.	THIRUNAVUKARASU	42	M	12	250	164	48	76.2	86	64	4	1.2	23	21	12	7	13.4	8	Multiple Velum oro	Velum, Oro	3
													12	19	5.4	8.6	6.8	9.7			
													16	15	11	12	10.5	11.6			
21.	SARASWATHY	29	F	51	58	132	22.9	83.5	86	65	3.6	0.8	12	13	0	0	1.3	1.2	Multiple Velum oro	Velum, Oro	3
													14.6	17.7	3.1	5.6	3.2	6.6			
													16	25.7	15.4	24.3	14.5	22.5			
22.	DEEPANRAJ	26	M	4	37	367	46.4	81.3	87	49	3.75	1.02	12	19	5.5	10	6.2	9.8	Multiple Velum oro EPI	Velum, Oro EPI	6
													20	7	0	0	1.5	1.3			
													18	17	6	0	5	0.5			
23.	KANDASAMY	49	M	59	34	239	43.7	59.3	95	88	3.9	1.06	12	21	0.3	0.7	1.2	1	Multiple Velum oro EPI	Velum, Oro EPI	6
													8	14	4	3	3.5	3			
													12	9	6	4.5	5.4	6			
24.	LOGANATHAN	36	M	4	12	102	27.9	73	91	81	2.5	1.4	10.5	11.6	11	9	13.2	10	Single epi	EPI	6
													14	6.9	13	5.8	14	7			
													14	24	3	2	5	6.3			
25.	NAGARAJAN	41	M	17	392	210	67	68.2	81	71	3.6	1.2	13	18	0	0	1.3	1.2	Multiple Velum EPI	Velum EPI	6
													13	9	9	9	10.5	10.3			
													16	31	0	0	1.2	1.4			
26.	MURUGAN	40	M	14	318	339	56.4	78.8	84	62	3.7	1.38	17	28	0	0	1.3	1.2	Multiple Velum oro	Velum, Oro	3
													19	15.8	6.9	11.5	7.4	13.5			
													15.3	34	14	31	16	34			
27.	RAJESH	33	M	4	8	177	15.7	74.9	97	95	2.5	0.8	13	17	12	15	13	16	No Obstruction	Velum	2
													14	10	12	8	11	9			
													18	25	17	23	16	21			
28.	SRINIVASAN	42	M	6	51	223	24.1	73.6	92	84	3.9	1.4	17.9	22.6	0	0	1.2	1.2	Single velum	velum	2
													15.3	9	11.2	7.2	10.5	6.8			
													15	19	12	15.4	14	16.4			
29.	RAVICHANDRAN	40	M	8	29	118	22.1	82.3	91	79	4.4	1.5	5.5	12	2	7	3	5	Multiple Velum oro	Velum, Oro	3
													9.9	13	4.5	7.5	3.8	6.8			
													12	17.7	10	13	11	12.3			
30.	SARASWATHY	38	F	34	28	117	25.4	72.5	92	85	5.2	1.2	12.9	20.4	0	0	1.2	1.4	Single velum	velum	2
													12.9	9	6	7.2	5.4	6.5			

S.NO	NAME	AGE	SEX	POLYSOMNOGRAPHY							DYNAMIC MRI									DISE	
				CENTRAL APNOEA	OBSTRUCTIVE APNOEA	HYPNOEA	AHI	MEAN HR	MEAN SPO2	MIN SPO2	SOFT PALATE DIMENSIONS		AIRWAY DIMENSIONS						LEVEL OF OBSTRUTION	LEVEL OF OBSTRUCTION	GRADING
											LENGTH	THICK	AWAKE		MULLER		SLEEP				
													AP	LAT	AP	LAT	AP	LAT			
													14	22.8	11.1	21.7	12	21.4			
31.	DINESH	32	M	61	219	582	79.2	101.8	74	48	4.2	1.1	8.1	10	4.5	3.4	4.3	3.6	Multiple Velum oro	Velum, Oro	3
													11	3.5	0	0	0	0			
													19	14	17	12	14	11.2			
32.	CHRISTY NIRMALA	43	F	3	15	142	17.8	83.8	96	80	3.6	1.3	13.1	13.5	5	5.5	6.4	7	Multiple Velum EPI	Velum EPI	6
													12.1	12.1	4.6	8.4	5.3	7.8			
													14.3	15.8	1.2	1.3	2.3	3.5			
33.	KRISHNAVENI	34	F	8	29	76	14.1	85.5	97	93	3.8	1	21	22	20.3	21.6	21.1	23.4	SINGLE TONGUE BASE	SINGLE TONGUE BASE	5
													17.6	19.8	9.6	7.3	10.6	6.4			
													21	37.4	7.4	35.2	9.6	25			
34.	ARUNA	41	F	12	102	121	32	81.3	92	86	4.4	1.2	3.7	8.9	0	0	1.3	0.7	Multiple velum and tongue base	Multiple velum,tongue base	5
													4.8	8.6	3.8	7.9	3.5	7.8			
													24.6	38	7	35	9.8	24.8			
35.	PREMA	40	F	4	12	112	27.9	73	93	81	5	1.3	16	24	14	16	15	14.5	Single tongue base	Single tongue base	5
													9.1	13.6	2.2	2.6	4.6	4.8			
													21	38	7	35	9.87	24			

MASTER CHART KEY

AHI	:	Apnea Hypopnea Index
OSA	:	Obstructive Sleep Apnea
DISE	:	Drug Induced Sleep Endoscopy
AP	:	Antero Posterior
LAT	:	Lateral
V	:	Velum
Oro	:	Oropharynx
Epi	:	Epiglottis
Min. SPO2	:	Minimum SPO2
Mean HR	:	Mean Heart Rate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INTRODUCTION
23
Obstructive sleep apnea syndrome (OSAS) is a component of sleep disordered breathing and this disorder 35 is characterized by excessive snoring and periodic apneas, hypopneas and arousals that leads to fragmented sleep in a repetitive specific duration .OSAS is a disease of modern ages and identified as distinct entity for past 20 to 25 years .At present ,OSAS 23 has been identified as a separate risk factor or an entity for increased susceptibility to stroke, myocardial infarction, cardiac arrhythmias, hypertension, dyslipidemia, insulin resistance and diabetes mellitus, depression, sexual dysfunction .Impairment of alertness also increases the risk of susceptible
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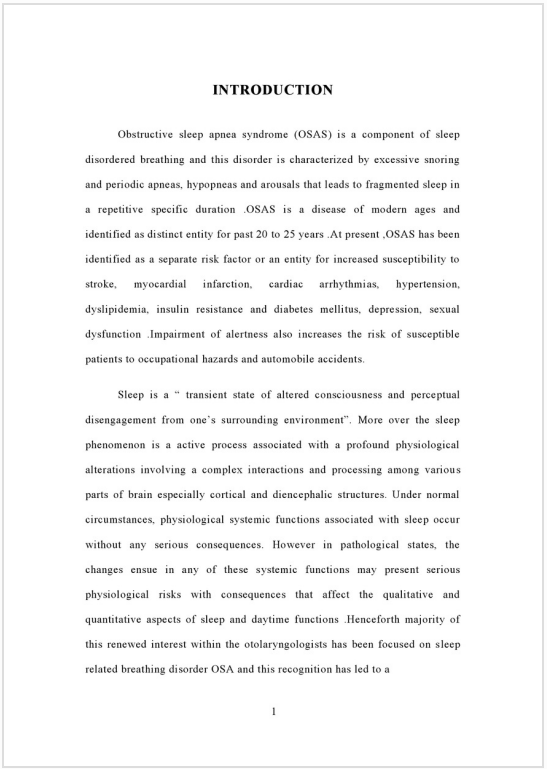

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a component of sleep disordered breathing and this disorder is characterized by excessive snoring and periodic apneas, hypopneas and arousals that leads to fragmented sleep in a repetitive specific duration .OSAS is a disease of modern ages and identified as distinct entity for past 20 to 25 years .At present ,OSAS has been identified as a separate risk factor or an entity for increased susceptibility to stroke, myocardial infarction, cardiac arrhythmias, hypertension, dyslipidemia, insulin resistance and diabetes mellitus, depression, sexual dysfunction .Impairment of alertness also increases the risk of susceptible patients to occupational hazards and automobile accidents.

Sleep is a " transient state of altered consciousness and perceptual disengagement from one's surrounding environment". More over the sleep phenomenon is a active process associated with a profound physiological alterations involving a complex interactions and processing among various parts of brain especially cortical and diencephalic structures. Under normal circumstances, physiological systemic functions associated with sleep occur without any serious consequences. However in pathological states, the changes ensue in any of these systemic functions may present serious physiological risks with consequences that affect the qualitative and quantitative aspects of sleep and daytime functions .Henceforth majority of this renewed interest within the otolaryngologists has been focused on sleep related breathing disorder OSA and this recognition has led to a

1